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THE CHICAGO MEDICAL SCHOOL
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SYMPOSIUM:

I. GASTRO-INTESTINAL EMERGENCIES

An Internist's Approach

MARTIN M. KIRSHEN, M.D., F.A.C.P.*

Many symptoms considered characteristic for diseases and emergencies of the gastro-intestinal tract may be produced by pathological changes in other organs. Conversely, pathological changes of the digestive system not infrequently give rise to signs and symptoms which seem to have no connection with this system.

For example, it is common knowledge that an acute myocardial infarction may begin with severe nausea, vomiting, and epigastric distress following a heavy meal. Precordial pain, with its characteristic radiation, and the changes in the electrocardiogram indicative of myocardial damage may be slow or late in their development and appearance. Occasionally, cases of this type are diagnosed and treated as perforated ulcers or other abdominal emergencies.

A dissecting aortic aneurysm extending into the abdominal aorta may produce a palpable abdominal mass, agonizing periumbilical pain, and rectal bleeding, a picture simulating intussusception, mesenteric thrombosis, or paralytic ileus. In some older, chronic cardiacs, a sudden failure may be initiated by vomiting and severe pain in the right upper quadrant, due to distention of Glisson's capsule of the liver.

Projectile vomiting which is difficult to combat and occasionally is associated with excruciating pain may be the first manifestation of a brain tumor or a tabetic crisis. Central pneumonia, localized in the right middle lobe, may mimic a ruptured appendix with lower quadrant pain, nausea, vomiting, toxemia, and even jaundice, especially in children. Often in these cases, no physical findings are obtained over the involved lung, and only the thought of such a possibility and subsequent x-ray studies may prevent a dangerous and unnecessary operation. In some of these cases, the abdominal symptoms may be of such severity as to completely detract one's attention from the main condition.

Another rather common situation which may confront the physician is that of a patient who is seen or has been admitted to the hospital as an emergency, with a diagnosis of diabetic acidosis. Besides the known symptoms and the usual laboratory findings characteristic for this condition, the main complaints are abdominal. There is severe nausea and vomiting, diffuse abdominal distention, pain, muscle guarding, lack of bowel sounds, fever, and leukocytosis—all signs of a gastro-intestinal catastrophe. Although the latter possibility has to be

*Associate Professor of Medicine, The Chicago Medical School; Attending Physician, The Mount Sinai Hospital of Chicago; Senior Attending Physician, Michael Reese Hospital.

ruled out, one must be aware of the fact that all of these symptoms may be produced by acidosis.

Acute pyelitis or pyelonephritis, especially in women in the second half of pregnancy, may produce the stormy picture of an abdominal emergency. The textbook signs of this condition, such as urinary frequency, tenderness of the flanks, and chills, are not always present. In such cases, only a careful study and a culture of the urine may lead one to the correct diagnosis.

Last, but not least, it is well to remember that practically all diseases of the female organs, especially those with an acute onset, are associated with gastro-intestinal symptoms of varying severity. Adnexitis, twisted ovarian cysts, pedunculated subserous fibroids, extra-uterine pregnancies, ruptured corpus luteum cysts, and endometriosis may produce abdominal pain, rigidity, occasionally a silent abdomen, and very frequently nausea and vomiting.

In the same manner that diseases of distant organs and structures may produce gastro-intestinal symptoms, diseases of the organs of the gastro-intestinal tract may manifest themselves by symptoms in adjacent or remote areas of the body. Substernal or precordial pain, produced by cardiospasm, esophageal ulcer, or cancer of the cardia and the night angina of an hiatus hernia are of common occurrence. Moreover, it is a known fact that the presence of "silent" stones in the gallbladder may be an important contributory factor in some cases of angina pectoris. Surgery in these cases may markedly improve or even eliminate the pain.

Penetrating ulcers of the posterior duodenal wall, not infrequently seen in older individuals, and carcinomas of the body and tail of the pancreas often cause unbearable back pain, simulating radiculitis or cord tumor, long before the characteristic symptoms of the original disease become manifest.

Acute pancreatitis is occasionally associated with severe precordial pain. To make the diagnosis of this condition even more difficult, electrocardiographic changes indicative of myocardial damage may be found. These changes may be due to the shock which is often encountered in hemorrhagic pancreatitis, or possibly to the excessive loss of potassium during the severe vomiting. Potassium concentration in the gastric juice is about three times as high as that in the serum.

Thrombosed and incarcerated hemorrhoids, severe proctitis, and indurated rectal fissures may become very distressing by reflexly producing pain in the thighs, calves, and even the soles of the feet. Proctologists see these conditions more often than do internists.

Metastases to the lung and bones with associated symptoms may occasionally be traced at the post-mortem table to a small, symptomless malignancy of the stomach or intestinal tract. Severe neuritis, cutaneous spiders, or esophageal bleeding may be seen as early manifestations of cirrhosis of the liver long before any other clinical evidence of the disease appears. Severe abdominal cramps in chronic lead poisoning or porphyria may test the diagnostic acumen of anyone.

Gastro-intestinal diseases, especially those of longer standing, share one peculiarity with chronic diseases of other organs. They occasionally produce systemic changes and symptoms long before specific, characteristic signs become apparent. For example, loss of weight, loss of appetite, subfebrile temperatures, weakness, and fatigue may be found in cases of chronic amebiasis without a history of diarrhea or abdominal pain. Similar symptoms are seen in carcinomas of the right colon, especially of the cecum, many months before a mass becomes palpable or interferes with the normal passage of the intestinal contents.

From all these examples, which could be multiplied many times, it becomes evident that gastro-intestinal diseases may manifest themselves not only by localized, but not infrequently also by distant and generalized symptoms. Conversely, pathological changes in other organs may mimic symptoms characteristic of changes in the gastro-intestinal tract. Therefore, the interpretation and proper allocation of these symptoms may be at times a difficult task in differential diagnostic considerations.

Gastro-intestinal emergencies, for the most part, are medical problems as long as the diagnosis has not been established, but the cooperation of an experienced surgeon is desirable even in the preoperative period.

Although exploratory laparotomies may at times be unavoidable, the importance of making a prompt and precise preoperative diagnosis in cases with acute abdominal pain cannot be over-emphasized. Incorrect diagnosis and a needless operation in an alleged abdominal emergency may be as disastrous for a patient as the postponement of a necessary surgical intervention. The choice of procedure in these cases requires not only a knowledge of patho-physiology and a meticulous physical examination, but also the entire diagnostic acumen of the physician and the intuitive mobilization of all his past experiences and observations. The urgency of the moment does not permit long delay for extensive laboratory work. Although such studies may be important and contributory to the diagnosis of abdominal emergencies, a skillfully taken history and an unhurried cross-examination of the patient will elicit important information. Snap diagnoses have no place in gastro-intestinal emergencies.

Occasionally, the symptomatology described by a patient may sound like a textbook description of a disease, but more often the history taking requires more than merely asking the patient about his immediate complaints. His former experience with similar attacks and his past medical record may be of utmost importance and a history of these must be obtained.

The physical examination must be of painstaking nature and not limited only to the abdomen because it is a known fact that diseases of the chest, the nervous and cardio-vascular systems, and the pelvic organs, as well as systemic and metabolic disturbances, may be the source of gastro-intestinal symptoms.

In the examination of the abdomen, attention should be paid to the presence of masses, a palpable liver and/or spleen, localized or generalized tenderness and rigidity, rebound abdominal pain, distention, and fluid in the abdominal cavity.

The presence or absence of bowel sounds and their quality, if present, should be determined by auscultation. To omit the examination of the inguinal, femoral, and obturator canals and the umbilicus may lead to grave errors.

No examination of a patient with a gastro-intestinal emergency is complete without a rectal and pelvic examination. When one remembers that 70% of the cancers of the digestive tract are found in the rectum and sigmoid and that a high percentage of them are accessible to the palpating finger, there is no need to emphasize the importance of these procedures. In older individuals, fecal impaction may occasionally be found to to be the cause of an intestinal obstruction. Tenderness in the vault of the rectal ampulla is frequently an additional sign of acute appendicitis. The finding of blood, tarry stools, pus, and mucus on the examining finger may also be of diagnostic significance. That a pelvic examination is a must, cannot be repeated often enough. Dire consequences may be the result of such an omission.

A scout film of the abdomen, best read by an experienced roentgenologist, often adds important clues to the diagnosis of abdominal emergencies.

According to the law of averages, there are five major pathologic conditions responsible for the most common gastro-intestinal emergencies. Therefore, we shall limit the discussion to these five diseases. They are: (1) acute appendicitis; (2) acute gallbladder diseases with or without stones; (3) perforation of peptic ulcers; (4) acute bowel obstructions due to a great variety of underlying pathology; and (5) acute pancreatitis.

In the first four conditions, only the diagnosis is the contribution of the internist; the treatment is predominantly surgical. In the fifth condition, the treatment in most of the cases should rest with the internist.

Acute Appendicitis

Acute appenditcitis is one of the most common gastro-intestinal emergencies. The early diagnosis and treatment are important factors in the management

of the condition. As a rule, a patient with acute appendicitis is taken ill suddenly with slight cramping pain over the entire abdomen which eventually localizes in the right lower quadrant at McBurney's point. However, if the cecum has a loose mesenteric attachment, the inflamed appendix may be situated deep in the pelvis and may not cause pain, outspoken tenderness, or rigidity of the abdomen. A rectal examination may elicit the characteristic tenderness of the right ampullary wall.

With a retrocecal appendix, the pain may be localized in the right flank or the right iliac crest may be extremely sensitive to pressure. On occasions, with gangrene of the tip of a retrocecal appendix, the area of pain may be so high as to mimic acute gallbladder disease.

The pain in acute appendicitis, although developing suddenly, is not very severe and is commonly followed, (not preceded) by vomiting. The nausea and vomiting are not as severe, as frequent, or as exhausting as in acute gallbladder diseases. The temperature elevation is moderate, as is the increase in the white count. It is a good clinical rule to suspect conditions other than appendicitis when fever over 101° F. and a white count over 20,000 are present.

The diagnosis of appendicitis is usually easy to make. Rarely, it may become difficult or impossible. Conditions like acute gastroenteritis, food poisoning (especially that due to staphylococcus infections), inflammation of the pelvic organs in women, renal colic, pyelitis, pyelonephritis, and lead colic may be confused with an acute inflammation of the appendix. Since lower quadrant pain is present in all of these conditions, the presence of other symptoms, their development and sequence, must be used as criteria for the differential diagnosis. The difficulty lies in deciding whether or not the condition requires surgery. When acute appendicitis cannot be ruled out with certainty, an operation will do no harm; whereas the failure to operate in time may result in perforation and peritonitis.

Gallbladder Diseases

The conditions most commonly responsible for pain in the right upper quadrant of the abdomen are the inflammatory lesions of the gallbladder which, for the sake of simplicity, may be classified as (1) acute cholecystitis (with or without hydrops), (2) suppurative cholecystitis, and (3) gangrene of the gallbladder. From the pathologic-anatomical point of view, they represent different stages of the same condition, in which the diseased gallbladder may or may not contain gallstones. It is believed today (and there is good experimental evidence for this) that chemical changes of the bile, obstructions of the cystic duct, and stones are the responsible etiologic factors. Infections, formerly considered so important, are rarely of any significance.

Diagnosis may not be difficult when the classical symptoms are present. However, as in other abdominal conditions, the differentiation between pleurisy, pneumonia of the right lower lobe, duodenal ulcer, appendicitis, pancreatitis, and renal colic must be made by a carefully taken history, by a diligent physical examination, and by the application of laboratory tests. An x-ray of the chest and a flat plate of the abdomen may occasionally be of great help.

An attack of acute cholecystitis begins with pain in the right upper quadrant, slight fever, and, occasionally, a chill. These symptoms may be mild in the beginning but in the presence of stones the pain may become colicky in nature and may be so agonizing as to make the patient double up or even go into shock. The pain frequently radiates to the back, to the right shoulder, to the neck, and to an area between the shoulder blades. The distended and swollen gallbladder is often palpable and very tender. Nausea and vomiting may be so severe and persistent as to exhaust the patient. The temperature is elevated, and may reach 101° to 104° in the first days. In one-third of the cases, a slight icterus is found and bilirubin and urobilinogen appear in the urine.

These symptoms may gradually disappear in a few days, but in some patients they persist and become more pronounced, indicating a progress of the disease. With the extension of the inflammatory infiltration of the gallbladder

wall, suppuration sets in and, eventually, gangrene and perforation may develop.

It is important to stress at this point that, in some cases, the pain, tenderness, and rigidity may not increase with the progression of the disease and that the symptoms, therefore, may not be indicative of the seriousness of the condition. More reliance should be placed on the increasing temperature, frequent chills, rapid pulse, toxemia, and rise in the leukocyte count. In some cases, it is a trying and responsible task to determine the proper time for surgical intervention. If perforation takes place, the picture is one of a sudden onset of generalized abdominal pain with all the signs and symptoms of acute peritonitis.

The treatment of an uncomplicated cholecystitis should be conservative, especially in the acute stage. Surgery is indicated in the presence of stones (in about 80% of cases) and, if possible, should be undertaken after a cooling off period of six to ten weeks. Severe cases with progressive symptoms should be operated upon without delay.

Perforated Peptic Ulcer

Perforation of a peptic ulcer is one of the most dramatic gastro-intestinal emergencies. The importance of early diagnosis cannot be over-emphasized. In no other condition are the chances of survival and recovery so dependent upon the recognition of the condition, irrespective of whether the ulcer is duodenal, gastric, acute, or chronic. Whatever the preceding compaints of these different types of ulcers may be, the symptoms of perforation are the same in all cases. Therefore, it is of no significance to identify the kind or site of an ulcer, but only to recognize that a perforation, with all of its consequences, has occurred. Heavy meals, large amounts of fluids, physical strain, and abdominal trauma (as in automobile accidents), may occasionally be precipitating factors.

The symptomatology of a perforated ulcer presents itself in three rather characteristic and successive stages. There is a sudden overwhelming pain in the upper abdomen which spreads to both subcostal areas and then extends over the entire abdomen. There is often localized tenderness in the right lower quadrant, simulating acute appendicitis, but due to the accumulation of spilled gastric contents in this area. Vomiting, occasionally with hematemesis, is an accompanying symptom and may lead one to suspect merely a bleeding ulcer. But the boardlike rigidity of the abdomen due to peritoneal irritation and the great tenderness in the epigastric area and in the gutter of the ascending colon are helpful clues in the differential diagnosis. Most of the patients are in shock, due to the excruciating pain; they are pale, cold, and clammy. The pulse is rapid and thready and there is a precipitous fall of the blood pressure. If at this stage the proper diagnosis is not made and the seriousness of the condition not recognized, a paradoxic amelioration in the condition of the patient sets in after a few hours.

The crushing pain diminishes in its intensity, the vomiting stops, the color and pulse of the patient improve, his anxiety vanishes and all seems to go well. This is the most deceiving phase in the course of a perforated ulcer. There is only one symptom left, and this may evade our attention—the persistent rigidity of the abdominal wall. If the patient is not operated upon in the next few hours, the final phase, with the picture of a generalized peritonitis, develops. The formerly retracted abdomen becomes distended, the temperature rises rapidly, the pulse becomes rapid and thready, and the patient appears gravely ill with a classical *facies hippocratica.*

In the differential diagnosis, gallbladder disease, renal colic, and appendicitis seldom offer difficulties. However, acute pancreatitis, mesenteric thrombosis, and, occasionally, referred pain from conditions outside the abdominal cavity, may simulate a perforated ulcer and lead to confusion. Although all these conditions may produce severe abdominal pain and shock, the boardlike rigidity of the abdomen is more characteristic of a ruptured viscus. A flat plate of the abdomen may show air under the diaphragm within twelve to twenty-four hours — in 50% of the cases. Therefore, negative x-ray findings do not rule out perforation.

Medical treatment consists of combating pain and treating shock, dehydration, and electrolyte disturbances in preparation for immediate surgery.

Acute Intestinal Obstruction

Another relatively frequent gastro-intestinal emergency is acute intestinal obstruction. Although there are few, if any, medical aspects to the treatment of this condition, it is usually seen first and diagnosed by the internist. Therefore, it is important to have a simple classification of the common causes of intestinal obstruction and to be aware of the most outstanding symptoms. A patient with colicky pain and vomiting is either developing or may have an obstruction. In no other abdominal emergency except perforation, will delay in diagnosis and treatment be accompanied by more dire consequences. Never let the sun set on a case of suspected acute intestinal obstruction!

The common causes of obstruction in infancy and in early childhood are anal malformations, intussusception, and volvulus. The most frequent lesions responsible for obstruction in middle life are hernias and post-operative adhesive bands. In advanced age, carcinomas of the colon and fecal impactions are the primary considerations.

The above mentioned and other less common local causes of bowel obstruction are grouped together as mechanical or dynamic ileus in contrast to the so-called adynamic, or paralytic variety of obstruction. This functional variey is produced by reflex inhibition of bowel motility via the celiac and mesenteric plexuses. It may be due to many causes of a diverse nature, such as peritoneal irritation from wounds, extravasation of blood, bile, pancreatic and stomach juices, toxemia, systemic infections, renal colic, lead poisoning, injuries of the spinal cord, fractures of the lower ribs, *etc.*

The symptomatology of acute intestinal obstruction is fairly characteristic, with pain, vomiting, cessation of bowel movements, and abdominal distention as the main features. The development of the clinical picture and the severity of the symptoms depend upon, and vary with, the site of obstruction. Nausea, vomiting, and continuous colicky pain are early and pronounced with obstruction of the small bowel. The higher the obstruction, the more violent are the symptoms, and the earlier is the development of dehydration, drop in blood pressure, peripheral vascular collapse, and shock. Alkalosis or acidosis develops, depending upon the amount and proportion of gastric and intestinal juice lost by vomiting or accumulated in the distended bowel loops. In most cases, blood potassium is high and ultimately, with the development of oliguria, extrarenal azotemia sets in, with a high N.P.N. and creatinine. In high intestinal obstruction, the abdominal distention is slight, visible peristalsis is often present and high pitched, and tinkling peristaltic sounds may be heard on auscultation.

In obstructions of the colon, the development of the above described symptoms is slow, gradual, and is preceded by a definite history of increasing constipation for a long period of time.

The diagnosis of acute intestinal obstruction is not difficult and can be made from the history, by a careful physical examination, and from the developing symptoms. A sudden or slow onset of colicky pain, nausea, vomiting, and abdominal distention should make one think of a bowel obstruction. The presence of an abdominal scar, a hernia, or a palpable mass in the abdomen and a pelvic or rectal examination may offer definite clues. X-ray evidence of a so-called stepladder pattern, with fluid levels in the bowel loops, is indicative of a small bowel obstruction. There is no objection to a carefully performed barium enema to demonstrate the site of a colonic obstruction.

The therapy of intestinal obstruction is mostly surgical, and the only duty of the internist may be the proper preparation of the patient for the procedure. The main features of this task are decompression by a Levin or Miller-Abbott tube, parenteral correction of the disturbed blood chemistry, and maintenance of the fluid balance.

Acute Pancreatitis

Acute pancreatitis is an abdominal emergency which is now known to occur

more frequently than was formerly believed. It is recognized today that there are two types of acute pancreatitis: acute pancreatic edema, and acute pancreatic necrosis. Both probably represent different stages of the same pathologic entity.

Although acute pancreatitis may occasionally be produced by trauma, infection, or circulatory disturbances, most of the cases are related to pathologic changes of the gallbladder and the biliary tract. These changes produce, by a mechanism not well understood, a functional disturbance of the sphincter of Oddi and an associated interference with and "deviation" of the flow of the pancreatic secretion.

The symptoms of acute pancreatitis may mimic not only other abdominal emergencies, but also thoracic emergencies. The onset is sudden, often a few hours after a heavy meal or an excessive intake of alcohol, and is characterized by an agonizing abdominal pain, frequently resistant to all narcotics. The pain may radiate from the epigastric region to the left or right hypochondrial areas, to the back, and to the shoulders. Occasionally, substernal radiation of the pain and changes in the electrocardiogram may falsely suggest a myocardial infarction. Pleural effusion in the left chest and areas of ecchymosis around the umbilicus and along the flanks (Gray-Turner sign) may be present in severe cases.

The diagnosis is difficult, and sometimes impossible, without laboratory information. A speedy amylase determination is of utmost importance when the disease is suspected. The elevation of amylase in the serum is of short duration, and should be looked for in the very early stages of the condition. The increase in lipase lasts longer, but is slower in development. A high antithrombin titer seems to be a new and promising test in the diagnosis of acute pancreatitis. Acidosis, sometimes hypochloremia with alkalosis, hyperglycemia, hypocalcemia, and glycosuria are additional laboratory findings in most of the severe cases. The development of a so-called lower nephron syndrome, with azotemia, is not uncommon.

It is agreed by all authorities that the treatment of acute pancreatitis should be medical, based on and directed by physiological considerations. Since the pathological changes of acute pancreatitis are due to a "deviation" of the pancreatic enzymes from their normal pathway, every effort should be made to restore this pathway, to relieve the obstruction or spasm of the sphincter of Oddi, to reduce the pancreatic secretion, and to avoid every known physiologic or pharmacologic stimulation of the gland. The external secretion of the pancreas is normally controlled by the vagi and by the hormone secretin, which is produced in the duodenum upon the entrance of the acid gastric contents. Therefore, all vagotonic drugs such as prostigmine, morphine and its derivatives, and Demerol should be avoided. All of these may also increase the spasm of the sphincter of Oddi. To prevent hydrochloric acid from entering the duodenum, oral feeding should be stopped and constant gastric suction should be instituted. Banthine, 100 mg. every six hours, may be of considerable help in reducing pancreatic secretion.

It is a difficult task to combat pain in acute pancreatitis since no morphine or Demerol should be used. Mild pains in acute pancreatic edema may respond well to nitroglycerine. In excruciating, severe pain, uni- or bilateral paravertebral block or 300 mg. Tetra-ethyl-ammonium chloride administered intravenously every six to eight hours have been used, with excellent results. Should the use of morphine become unavoidable, large doses of atropine or Banthine should be administered simultaneously.

The intravenous administration of fluid should be guided not only by frequent determinations of electrolytes, but also by a consideration of the action of some of the fluids on pancreatic secretion. Hypertonic glucose solutions and amino-acids administered intravenously may produce profuse pancreatic secretion and should therefore be avoided. Plasma and saline solutions may be used without any harm. Insulin, which is used to combat an occasional hyperglycemia in acute

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II. THE ACUTE ABDOMEN

A Surgeon's Approach

EMANUEL MARCUS, M.D., Ph.D.*

Contents

Introduction

When symptoms tolerable to the abdomen are severe or appear with relative suddenness, or both, they are alarming to the patient and may be perplexing to the doctor. The presenting complaints may be so characteristic and preliminary examination so clear-cut that the clinician, be he surgeon, general practitioner, internist, can immediately decide whether the disease be amenable to medical means or requires surgical therapy. Where the presenting complaints do not fit any pattern and the examination confuses rather than clarifies, the differential diagnosis between conditions that require surgical intervention and those which either do not require it or should not have it becomes equivocal. In such cases, it becomes the responsibility of the surgeon to make the decision, using all information available from the patient, from his own faculties of perception, and from the laboratory. This responsibility demands conscientiousness, integrity, and clarity of thought. As the history is taken and the examination made the surgeon must consider which diseases are most probable, statistically, with reference to age, sex, factors in the history, and presenting symptomatology. Is this disease amenable to surgical treatment? Is it immediately threatening the patient's life? What are the possible conditions that could produce his symptom complex in which surgical intervention is not necessary, or indeed would be a disservice?

Under such circumstances the surgeon must use his judgment, a summation of all evidence, clinical and laboratory, project these onto the background of his experience and knowledge, and make his decision. Such decision encompasses not only a differential diagnosis but also a plan of treatment, not the least of which is the question of timing of the surgical intervention — whether it must be immediate or more properly delayed for further diagnosis or all-important pre-operative management.

A. Perforations or Threatened Perforations of Hollow Viscera — Non-traumatic

1. Inflammatory

Acute Appendicitis

Acute appendicitis can be the easiest diagnosis imaginable, and upon occasion the most difficult. The characteristic story of mid-abdominal or peri-umbilical discomfort or of early nausea, followed by shift of discomfort to a pain in the right lower quadrant within 24 hours, and usually accompanied by anorexia, immediately suggests appendiceal disease. Constipation is more commonly found than diarrhea. Fever is not characteristic of early appendicitis in the ob-

*Assistant Professor of Clinical Surgery, The Chicago Medical School.

structive phase, but rather is found late, when the peritoneum is involved or the blood supply compromised. This is the usual history given within the early hours — except at both age extremes.

Physical examination is usually less characteristic than the spontaneous symptoms, for the latter are determined by the innervation of the ileocecal area, which is quite constant. However, the signs on examination will depend on the actual location of the diseased organ and the structures involved in the localized peritoneal irritation. Thus, the characteristic picture of maximal pain to pressure over McBurney's point, rebound tenderness sharply localized in the right lower quadrant, and a positive Rovsing sign are not diagnostic of appendicitis, but of a localized peritonitis at McBurney's point. These conditions obtain when the tip of the inflamed appendix touches the peritoneum of the anterior abdominal wall at a point on a line between the umbilicus and the anterior superior iliac crest. However, the tip of the appendix or the inflamed portion of the appendix may lie on the posterior abdominal peritoneum, on the ureter, against the gallbladder, on the urinary bladder, on the sigmoid colon or rectum, or on a fallopian tube. The signs on examination, then, will be those referable to the organs in proximity to the diseased tissue. It is not surprising, therefore, that a patient may have flank pain, frequency, pelvic discomfort, rectal pain, or pain simulating biliary colic — and still have a good story compatible with acute appendicitis. Probably as high as 30 per cent of all appendices are wholly or partially retrocolic. Rectal or vaginal examination will sometimes aid in revealing its position. The iliopsoas or obturator signs are designed for the same purpose—the location of the diseased appendix, not the diagnosis of appendicitis. Laboratory data may be corroborative but are also not, in and of themselves, diagnostic. A high percentage of polymorphonuclear neutrophils is more characteristic than a high total white-cell count. Occasionally, when the clinical impression is acute appendicitis, the white count may show no change from normal values. In such cases, a gangrenous appendix is usually found, representing a rapid, fulminating process to which the body has not had time to mobilize its defenses.

Acute Cholecystitis

Characteristically, this disease occurs in the rather obese female who has had some pregnancies and the physiologic hypercholesterolemia that comes with them. Obviously, there are other causes of disturbed cholesterol metabolism; therefore the disease also occurs in other types of individuals. Frequently, cholecystitis with stones occurs without apparent metabolic disorder in the very young and the aged of both sexes, providing the exception to the rule.

The most frequently encountered symptom complex consists of postcibal bloating and belching, pain under the right costal margin referred to the somatic nerves of that area, selective dyspepsia to fried or fatty foods, and the symptoms of pylorospasm. The local signs are those of peritoneal inflammation at the anatomic site of the gallbladder, differing from appendicitis, therefore, in location only. The systemic signs are those of infection — fever and leukocytosis. A flat plate of the abdomen may reveal radioopacities in the gallbladder.

If the cholecystitis is accompanied by formation of stones, the pain may be of a colicky nature as the calculi attempt to pass through the cystic duct. When the cystic duct is obstructed and the gallbladder mucosa continues to secret mucus, the gallbladder will become distended and the pain will be boring and deep. The pain of distention, as occurs when any lumen is obstructed, and the superimposed edema and infection may set the stage for compromise of the blood supply, resulting in a gangrenous wall. Under such conditions the local peritonitis will be great and, if the defenses of the individual are weak, a generalized peritonitis may occur.

The question of treatment of acute cholecystitis by early or delayed surgical therapy is not properly to be discussed here, for the valid reasons on both sides of the argument are largely technical. A word about medication for relief of the acute symptoms, however, may be

well taken. Morphine should not be used for the pain of biliary colics (or other colics) for it raises pressure in the biliary system through constriction of sphincters and by increase in smooth muscle tone. Atropine and nitroglycerin are more effective, especially in combination with a narcotic that is also a muscle relaxant, such as meperidine.

Myocardial disease and diaphragmatic pathology are always to be excluded in the diagnosis of acute cholecystitis, as will be emphasized later.

Acute Pancreatitis

The exact etiology of pancreatitis is not known. However, some of the predisposing factors are discerned from the type of patients that it claims. No distinction will be made between edematous pancreatitis and hemorrhagic pancreatitis since the difference is apparently a quantitative one. Acute edematous inflammation of the pancreas usually subsides within a few days, whereas the fulminating hemorrhagic cases may progress to extensive abdominal fat necrosis.

The patient is usually, though not necessarily, one with a history of some biliary disease. Most frequently, the attack follows dietary indiscretion, either alcohol or a very large meal, or both. Pain is of rather sudden onset and is very severe. It is usually a through and through pain — epigastrium to back. The patient finds it difficult to remain in one position, especially in the supine. Rather, he moves about and is usually found doubled over in the sitting position. Vomiting is common.

Findings include a degree of shock out of proportion to other findings, a shock-like pulse, a very tender epigastrium exhibiting the rebound phenomenon, and reflex ileus. The blood amylase is found to be elevated during the first 24 to 48 hours of the disease, but normal readings do not rule out the disease. Blood lipase elevation is thought to be even more reliable. Both enzymes are present in increased amounts in the urine. When fat necrosis is extensive, hypocalcemic tetany may be present due to the binding of free calcium ions by fatty acid radicals of the digested fat.

Diverticulitis

Diverticula of the lower colon are common; they are outpouchings of the mucous membrane of the bowel through points in the intestinal wall made weak by the passage of blood vessels through them. Inflammatory disease of these diverticula is associated with local peritoneal irritation, spasm of the bowel locally, threatened or actual perforation, formation of an abscess mass, and even obstruction. The patient, usually middle aged or older, presents himself with complaints that are both general and local; the general ones are those of infection, the local ones are those of pain in the lower abdomen, tenderness, and constipation with cramps. The symptoms are usually not new but rather recently progressive. Examination reveals tenderness with rebound phenomenon in the lower abdomen, usually on the left, and possibly a palpable mass. Sigmoidoscopic examination will reveal spasm but no mucosal lesions. It is frequently impossible to differentiate diverticulitis of the lower colon from malignancy. This is true even with the lesion in the hand at operation. Of course, diverticulitis will respond better with conservative therapy than will malignancy. When a mass is present, surgery is indicated, either to relieve the obstruction or to make a diagnosis, or both.

Salpingitis

Acute salpingitis is not seen as frequently as before the days of antibiotics. It must still be considered in the young female when the presenting story is not characteristic of other causes of low abdominal peritonitis. Pelvic examination may aid in the diagnosis. When found at exploration, the treatment is not operative but medical.

Meckel's Diverticulitis

A Meckel's diverticulum may undergo the same disease process that an appendix does. It can produce the same symptoms and frequently is confused with appendicitis. Meckel's diverticulitis is usually not diagnosed pre-operatively unless there is evidence that it has been bleeding. The bleeding is usually due to peptic digestion of the diverticulum resulting

from the heterotopic gastric mucosa (producing hydrochloric acid) in the Meckel's pouch.

Regional Enteritis

Regional enteritis is a granulomatous disease of small bowel that is usually insidious with a progressive story of cramps, diarrhea, and malnutrition. X-ray studies frequently reveal the first clue to the disease, a feathering of the intestinal mucosa or, later, an encroachment upon the lumen by the granuloma (the "string sign"). Occasionally, however, the disease first drives the patient to the doctor with severe right lower quadrant pain. The abdomen is opened and a characteristic picture is found of many matted loops of bowel with fistulae between the loops. Very careful questioning of the patient probably would have disclosed, not a 48 hour history, but a history of cramps, pain, and diarrhea of many weeks' or months' duration.

2.. Neoplastic

A carcinoma of the intestine may cause a perforation into the free peritoneal cavity. The site most frequently involved is the sigmoid colon. The perforation may be at the site of the carcinoma or, if the lesion is completely obstructive, a closed loop obstruction of the colon may cause the bowel to perforate at the point of greatest distention, the cecum. A typical story is that of an elderly patient who comes in with a story of progressive constipation and obstipation of days to weeks, with sudden acute pain in the right lower quadrant of the abdomen, and whose abdomen is found to be greatly distended and to have evidence of peritonitis. Such a patient usually has a malignancy of the sigmoid with perforation of the cecum.

3.. Other Perforations

Peptic Ulcer

Peptic disease is far and away most common in males. Sudden severe upper abdominal pain in a patient with a known ulcer history whose abdomen is rigid in the right upper quadrant offers no difficulty in diagnosis. The chemical peritonitis will produce an ileus, with rapid loss of bowel sounds. Free air in the peritoneal cavity will be demonstrable in about half of such cases. Not infrequently, the first evidence of peptic disease will be the accident of perforation; this is particularly true in the young and old. Although most commonly a disease of the third, fourth, and fifth decades, peptic ulcer may be found in the first days of life and in the seventh decade of life. Statistically, this disease appears to be becoming ever more important. Even when no previous diagnosis has existed, careful history taking may elicit recent symptoms of pyrosis, a chemical type of epigastric pain when the stomach is empty, or a taste of blood which some who bleed into the gastrointestinal tract can reliably report.

Early following perforation the above symptomatology is characteristic. Later, it may be difficult to differentiate a perforated ulcer from an appendiceal rupture or cecal pathology, because of the leakage of gastric contents down the right paracolic gutter.

A *forme fruste* perforation is one that is quickly walled off, usually by the pancreas. Since this rupture is usually posterior, the anterior abdominal wall signs are minimal and back pain with other signs of pancreatitis may predominate.

Ruptured Tubal Pregnancy

Sudden syncope with advancing shock in a female in the reproductive age whose abdomen is painful and exhibits rebound tenderness should be considered a ruptured ectopic pregnancy until disproved. Usually, there will already have been a diagnosis of pregnancy or the past menstrual period or two will have been abnormal. It will ordinarily be difficult to maintain her blood pressure, even with transfusions, until the bleeding tube is repaired. All peritoneal signs are due to blood in the peritoneal cavity, the general signs to hemorrhagic shock.

B. Traumatic Perforations of Hollow Viscera, Fractures of Solid Viscera, and Tears of the Mesentery — Traumatic

Hollow Viscera

Penetrating and perforating wounds of tbe abdomen must always be explored.

The site of the stab wound or the trajectory of a missile will give the surgeon a clue as to which organs are involved, but all hollow viscera will almost always have to be inspected. Traumatic wounds are more serious the lower in the intestinal tract they occur. Wounds of the buttocks and hips may injure the bladder and the rectum; wounds of the thorax may injure upper abdominal viscera.

Blunt force to the anterior abdominal wall may also cause perforation of a hollow viscus, especially if the viscus is distended with fluid and air.

Spleen and Liver

These solid organs are frequently injured by blunt force applied to the parietes. A ruptured spleen will usually give a picture of profound shock with the signs of an increasing amount of blood in the coelomic cavity. Not infrequently, the accident will have occurred hours, days, or even weeks before, with apparent recovery, followed by a sudden recurrence of all symptoms. This is the so-called delayed splenic hemorrhage. The original crack in the spleen was sealed with a wedge of clot which became squeezed out at the later date, resulting in the new picture of hemorrhagic shock and blood in the abdominal cavity. Fracture of the spleen must not be forgotten in trauma to the left lower thoracic cage.

Many instances of small lacerations of the liver will heal spontaneously without operative intervention. Crushing upper abdominal injuries may split the dome of the liver, resulting in massive spillage of blood and bile, requiring immediate operative effort.

Mesentery

Occasionally a patient is brought into the emergency room with a history of having fallen from a height and having landed on his feet. As a rule, there will be fractures of the ankle region, including those of the calcanei. The patient may be in profound shock, the reason for which may not become apparent if all attention is directed to the extremities and none to the abdomen. Turning one's attention to the abdomen will occasionally disclose unmistakable signs of blood in the peritoneal cavity. Following vigorous supportive therapy, immediate celiotomy (or unfortunately, if not immediate enough — autopsy) will disclose the source of hemorrhage to be the detachment of the mesentery of the small bowel from its dorsal root. This is the result of a shearing action exerted when the patient landed on the ground, the weight of the intestines continuing downward tearing the mesentery and its vessels. Bowel resection is almost always required.

C. Gastro-intestinal Bleeding

1. High

Peptic ulcer is the most common clinical cause of massive upper intestinal bleeding. Esophageal varices due to portal hypertension and lesions of the stomach, malignant and benign, follow in frequency. When a patient is brought in with evidence of vomiting blood and is in incipient or profound shock, the problem at hand is not one of diagnosis, but of support. Almost always, the definitive diagnosis can wait until the patient is revived—which may be a matter of hours or days. Usually there will be an antecedent history pointing to peptic disease or to portal hypertension. There may be a scar of previous surgery for ulcer, a large liver with evidence of collateral circulation for bleeding varices.

In the case of the patient over 50 who does not maintain the elevation of blood pressure produced by transfusion, who does show evidence of arteriosclerosis in the peripheral arteries, definitive therapy may precede diagnosis or, if the diagnosis is ulcer disease, it may be assumed that the sclerotic vessel that is bleeding cannot contract. Under such circumstances operative intervention may be more successful if carried out immediately. If, from history and findings, the most likely diagnosis points to bleeding esophageal veins, the immediate insertion of an esophageal compression balloon is indicated.

2. Low

Massive lower intestinal bleeding also requires immediate supportive therapy and then diagnosis for definitive treatment. Lesions of the left colon will be

the chief causes of such hemorrhage. Carcinoma, benign polyps, diverticulitis, and internal hemorrhoids will be the main culprits. Visualization per endoscopic means and/or biopsy will be followed as soon as is feasible by roentgen studies. In children, bleeding from peptic ulceration of a Meckel's diverticulum is to be remembered. Bright blood can be traced to small intestinal bleeding (polyps, tumors) if there is an associated hyperperistalsis and rapid progress of intestinal contents. Even duodenal ulcers have produced bright bleeding per rectum, but this is not the rule.

D. Obstructive Causes (Including Colics)

Obstruction to the passage of contents in the gastro-intestinal tract in a caudal direction will produce acute symptoms. If the obstruction is high in the tract, vomiting will predominate; if low, distention of the proximal bowel (and of the abdomen) will be prominent. In between the very high and the very low there will be varying combinations of both. This potentially serious situation will be recognized by symptoms localized to the abdomen and by others related to the general body economy.

The paralytic type will be recognized as a generalized paralysis of all sections of the gut, usually following recent surgery, accompanying a peritonitis, or consequent upon disease elsewhere (*e.g.*, renal colic, fractures of pelvis, vertebrae, and ribs, or pneumonia). There will be no bowel sounds, a uniformly distended abdomen with no specific tender area, and usually some antecedent or concomitant disease.

The mechanical type may be recognized by the hyperperistaltic sounds which become metallic or tinkling in character and then fade out completely as the bowel musculature becomes exhausted. The most common causes are herniae of various kinds and old (fibrous) adhesions from previous surgery. External herniae and scars on the abdomen are to be looked for. In the newborn, malrotation of the gut and stenoses or atresias are to be considered. Volvulus of the sigmoid, in the severely constipated, is to be thought of. Intussusception, especially in children, is to be remembered. Obstruction of the duodeum by a tight superior mesenteric artery is occasionally found in the infant. Tumors, both primary and metastatic, must be considered in every case as possibilities until ruled out. Roentgenography may assist in all cases of mechanical obstruction by showing sudden cessation of downward progress of gas in the bowel.

Of great moment in assessing an acute abdomen with a tentative diagnosis of mechanical intestinal obstruction is the all-important question, "Is this obstruction simple, or is it strangulated?" A strangulated obstruction is one in which the blood supply to a portion of the gut has been compromised; it demands immediate surgical intervention. Strangulation will ordinarily produce an area on the abdomen more sore and tender than the rest of the abdomen; in addition, the patient will be sicker than another without strangulation. The general disturbance of body economy is mainly that of fluid and electrolyte loss into the lumen of the intestine and may be sufficent to kill the patient, in and of itself, if not corrected in the pre-operative phase of treatment.

Colics may be included with obstructions. Colic of the cystic duct and of the appendix have already been mentioned. Colic of the urinary tract, in addition to urinary findings, may cause reflex findings in the abdomen, such as reflex ileus with distention. Similar reflex ileus with distention of the abdomen may have causes other than renal colic, *e.g.* — blood around the kidney or in the retroperitoneal space, traumatic or otherwise.

E. Vascular

Mesenteric Thrombosis and Embolism

Infection anywhere in the intestinal tract or its derivatives may lead to ascension of the inflammatory process centrally, resulting in a thrombophlebitis. This process may lead to a localized phlebitis in the veins of a small segment of gut or may spread to many venous radicles and to the portal vein itself — pylephlebitis — with retrograde spread to all veins that subtend it. The bowel so

affected may become blood-logged, with necrosis, seepage, and peritonitis. The clinical picture is similar to that of strangulated obstruction with peritonitis. The general picture is one of infection and shock. The most common clinical antecedent is appendicitis.

Arterial thrombosis can occur in mesenteric arteries as well as in the vessels to the toes, the kidney, or the heart. It is a disease of the aged and those known to have severe atherosclerosis.

Mesenteric arterial embolism is a sudden and spectacular abdominal catastrophe. It is most commonly seen in patients who have recently sustained a myocardial infarction and now have developed a ventricular mural thrombus. Such patents rapidly develop an acute abdomen and are thoroughly sick. The antecedent history is all-important. The infarcted intestine will be dry and greenish-grey; seepage of intestinal contents is rapid. Even in these very critically ill patients resection of the infarcted intestine is mandatory.

Infarction of the spleen on the same basis (embolus to splenic artery) can usually be overcome by the patient. This is unlike venous thrombosis which will result in splenic rupture and intraperitoneal hemorrhage or gastric hemorrhage from over-distended vasa brevia and coronary veins.

Twisting of Vascular Pedicles

Any structure on a pedicle can be infarcted by obstruction of its arterial supply or distended with blood to bursting if only its venous drainage is obstructed. Twisting of an ovary is the most commonly occurring example. It is to be remembered that the entire blood supply of the intestines is really a pedicle, since the root of the mesentery is really only about four inches long, whereas the gut it supplies is about twenty feet long. Volvulus, therefore, can be simply obstructive of the bowel lumen or, at its worst, can strangulate the intestinal blood supply by twisting the intestinal pedicle — the root of the mesentery.

III. Diseases to be Considered and Ruled Out as Not Amenable to Surgical Therapy

A. Cardiac
   1. Myocardial infarction
   2. Acute pericarditis

B. Pulmonary
   1. Pneumonia
   2. Diaphragmatic pleurisy
   3. Embolus

C. Gastric
   1 Acute gastro-enteritis, including alcoholism
   2. Tabetic crisis

D. Vascular
   1. Dissecting aneurysm
   2. Fusiform aneurysm

E. Allergic
   1. Periarteritis nodosa (?)
   2. Abdominal allergy, including Henoch's purpura

F. Hepatic
   1. Hepatitis
   2. Acute cardiac failure with hepatomegaly

G. General
   1. Porphyria
   2. Diabetic impending coma
   3. Hysteria

III. THE ACUTE ABDOMEN

A Gynecologist's Approach

M. WILLIAM RUBENSTEIN, M.D.*

The accurate diagnosis of the acute abdomen in the female must take into consideration those causes that are gynecological in origin. It becomes apparent that: 1) a careful and detailed menstrual history must be obtained; 2) a careful examination—including a pelvic examination, vagino-abdominal, recto-abdominal, and often recto-vaginal—must be made; 3) intelligent assistance of pertinent laboratory aids should be obtained; and that 4) a distended urinary bladder must be emptied, in order to avoid mistaking it for gynecological pathology.

In spite of these precautions, diagnostic difficulties do arise. Indeed, as in the acute abdomen due to other causes, especially in women of menstrual age, an accurate diagnosis may not be possible in certain cases until an exploratory laparotomy is done. Nevertheless, one must always attempt to arrive at a preoperative diagnosis for there are certain acute gynecological conditions that call for surgical intervention, while others do not. However, if one feels that he is dealing with a surgical abdomen, there should be no delay in operating.

The more important pathological conditions that affect the female reproductive organs and produce acute symptoms of possible surgical significance are as follows:

Ectopic Pregnancy

One of the most frequent and perhaps most important cause of all tubal pathology that leads to a surgical abdomen is ectopic pregnancy. The pathologic nature of ectopic gestation is such that its accompanying disturbance of abortion and/or rupture are common and wrought with great danger to the patient.

There seemingly have been more ectopic pregnancies within the last several years. There is no single explanation to account for this. However, with the advent and use of antibiotics, fewer occluded tubes have resulted following infection. Perhaps the tubes, though recanalized after a severe pyosalpinx, for example, may be left with some scarring, narrowing, and kinking of the lumina. Such conditions may allow fertilization, but also may interfere with proper migration of the ovum into the uterine cavity, thus allowing the conceptus to implant on the endosalpinx.

Because the tubal structure is not primarily suited or adaptable to pregnancy, the developing embryo has a complicated future. The growing conceptus will distend and stretch the tube and will, of course, burrow into its wall. It may, in this process, erode a blood vessel, break through the wall (rupture), detach from its implantation (abort), or bleed into its developing sac. This brief description helps in understanding the bizarre and variable symptoms that can arise. The aborted conceptus can be extruded into the free peritoneal cavity with no special difficulty or bleeding and may continue to grow as an abdominal pregnancy. The abortion may occur into the tube where it will become enclosed and later absorbed—or it may be forced toward and into the uterus. Various stages of bleeding can result during such changes, from minimal to profuse hemorrhage. When the changes such as detachment, rupture, or bleeding occur suddenly, some degree of symptoms, such as pain, shock, anoxia, *etc.*, will be present and indeed may be overwhelming.

Acute symptoms usually result from a sudden rupture of the tube or sudden tubal abortion. The symptoms of rupture are usually more severe than those resulting from tubal abortion. Only about 15% of the patients with ectopic pregnancies will present themselves in acute shock. In the main, this is caused by tubal rupture. The onset may be with sudden, severe lower abdominal pain, ac-

*Instructor, Department of Obstetrics and Gynecology, The Chicago Medical School.

companied by fainting and the associated signs of hemoperitoneum. In general, shock is proportional to the degree of bleeding and to the suddenness of the bleeding (even though not great in amount — 50 to 150 cc.). The escaped blood produces a painful chemical peritonitis. Sometimes there is pain in the region of the shoulder when the blood gravitates upward to cause diaphragmatic irritation.

The patient is very uncomfortable and restless, is usually doubled up, has her hand on her lower abdomen, "looks sick," and may get worse "right before your eyes." On abdominal examination, tenderness is pronounced and guarding and reflex rigidity are present. The patient has pain in both lower quadrants, if bleeding is at all extensive. However, it is much more pronounced on the affected side and may localize to one of the adnexal areas. On vaginal examination, the fornices may be tender and the cul-de-sac full. Fullness or a mass may be made out on the involved side. If the peritoneal irritation is very great, the bimanual examination may be very difficult, but a "suggestion" of increased pain and a feeling of fullness are often obtained. The fullness in the fornices, especially the posterior, is proportional, as a rule, to the amount of internal hemorrhage present.

The uterus may be normal in size or slightly enlarged, and may even feel softened. There may be no blood or a trickle of blood about the cervix or in the vaginal vault. In checking on the history, the patient usually relates that her last menstrual period was different—it was a little late or started on time, but in either case was light and spotty in character and then stopped or continued that way until the presenting acute symptoms befell her. There is usually some abnormality in the menstrual history. If the amenorrhea and spotting were prolonged (several weeks), fullness in the breasts, with possible other early changes of pregnancy, may also be present.

The question will often arise as to the differentiation between the patient with an incomplete uterine abortion with some concealed hemorrhage and in shock from one with a ruptured ectopic pregnancy. In the former, the presence of the soft enlarged uterus and absence of a lateral mass or fullness are helpful. In ruptured ectopic pregnancy, movement of the cervix causes pain, as a rule, (due to the irritation of the peritoneum by blood). In intra-uterine pregnancy that is threatening to abort or is in incomplete abortion, this practically never occurs.

In 85% of cases, however, the symptoms are subacute. It is in these patients that the diagnosis will call for the greatest of skill on the part of the doctor. "The doctor must be ectopic-minded." The patient is not acutely ill. She usually has two main complaints: 1) that her last period was peculiar—it "started sort of funny," was light, of a spotting nature and/or was late a few days and lasted off and on for a few days or perhaps a few weeks; 2) there is some abdominal pain or discomfort, sometimes sharp and colicky; perhaps not constant, but enough to make her think about it and even consult a physician. If the patient is very discerning, she will be able to state that the abdominal distress is localized on one side and in one of the lower quadrants. Usually spotting precedes the pain by several days. The patient may be obviously quite pale — a clue to "bleeding from somewhere"—and yet not be in shock or in any degree of discomfort. Here again, be "ectopic-minded."

The amenorrhea of several days or weeks may be classically present but its absence should not deter the diagnostician from considering ectopic pregnancy. Abdominal tenderness is usually less marked than in acute appendicitis and if on the right side, is situated below and medial to McBurney's point. Guarding and rigidity are especially present shortly after rupture—if there is peritoneal irritation. A palpable adnexal mass or fullness is present in about 20 to 70% of cases and is, therefore, in itself not necessarily of differential diagnostic significance. Pregnancy tests are of value only if positive. A positive hormonal pregnancy test means only that live chorionic villi are still present, allowing the chori-

onic gonadotropins to gain access to the maternal circulation. On the other hand, a corpus luteum cyst may also give a positive test and may be indeed confusing when associated with amenorrhea. Observation for some time is justifiable in order to get blood count comparisons and to await the results of a pregnancy test and other tests, provided that the patient is in a hospital and is very carefully watched. At the first signs of a change in the patient's condition, such as increased pain or the symptoms of internal bleeding, leading to a diagnosis of ectopic pregnancy, surgical intervention should be immediately considered.

It may be stated at this point that the use of either colpotomy or cul-de-sac needle puncture, or both, is very helpful. Liquefied — not clotting — blood, if obtained, is very helpful. Fresh, bright red blood is quite suggestive of active bleeding. This may be mixed with the darkened, old blood. It might be advisable to visualize the adnexal regions on colpotomy — a very useful measure. Culdoscopy in certain cases is also of great diagnostic aid. Perhaps the greatest benefit from needle puncture comes in the case that presents a bulge or fullness in the posterior fornix.

Sufficient blood and intravenous fluids should be given. If the patient is in shock (due to bleeding), blood should be started and surgery begun almost immediately. When the patient is especially bled out, anoxic, and in poor shape, the choice of anesthetic becomes an important point. Depressant drugs, which in themselves potentiate shock, should be avoided (such agents are I.V. pentothal, rectal anesthesia, and spinal anesthesia). In principle, one does not want to use a drug whose action cannot be easily counteracted.

The anesthetic of choice is probably cyclopropane because of the large proportion of oxygen which can be used and the peripheral vasoconstrictor effect associated with its administration. This, in itself, helps to counteract the peripheral vasodilatation present in shock. Ether is always reliable and is also safe in those cases where bleeding has been extensive.

Hemorrhage from the Ovary

The bleeding ovary can produce abdominal symptoms that must be differentiated from acute appendicitis, ruptured ectopic pregnancy, twisted ovarian cyst, and pelvic inflammatory disease. Bleeding usually occurs from rupture either of a Graffian follicle ("mittelschmerz"), corpus hemorrhagicum, or corpus luteum of the ovary. It may produce one of the most confusing of acute abdominal conditions. The pain may be severe, is practically always sudden in onset, and is in one of the lower quadrants, usually the right (about 15 to to 1). It is more often incorrectly diagnosed as acute appendicitis than any other acute condition in the abdomen with the exception of Meckel's diverticulum. It usually occurs in the middle to the latter half of the menstrual cycle—5 to 16 days before the expected next period. This is one of the most important points in the history for diagnosis. The onset, occasionally, may even follow the trauma associated with intercourse. Women under 25 years of age are most often affected. Frequently there is a history of previous attacks. The pain and tenderness may be as intensive as that of acute appendicitis and are usually about an inch or more below McBurney's point. The pain is most often on the affected side, but may be bilateral, and then later may become generalized or may radiate to the right flank, the lumbar area of the back, the right hip, or under the diaphragm, with pain in the right shoulder. Adnexal tenderness is present in about half of the cases and only occasionally is a mass felt (usually a ruptured corpus luteum cyst) on the affected side. (Here ectopic pregnancy must be closely differentiated.) These patients' symptoms may improve in 24 hours if hemorrhage has not been extensive, although some soreness and tenderness in the region of the ovary remains for three or four days. The acute symptoms may be severe enough to warrant surgical treatment in only about 6% of the cases. The amount of bloody fluid in the peritoneal cavity is usually small, but large amounts may also be found. The patient may even be admitted in semi-shock, with signs of

marked intra-abdominal hemorrhage, and at surgery only a ruptured follicle cyst may be found. Operative procedure may require only suturing, or expression of a clot and suturing of the bleeding area. Oophorectomy is not at all indicated.

Acute Salpingitis

Acute salpingitis is usually gonorrheal in nature. It starts, as a rule, somewhat gradually with chills and high fever. The patient may be quite sick and may be nauseated or even vomit. Vaginal discharge is usually present. Smears from the cervix and/or Skene's glands are frequently positive for the gonococcus. The pain is practically always bilateral, radiating across the lower abdomen. Vaginal examination causes pain when the cervix is pushed upward or if gently moved from side to side. The adnexae are acutely tender and there may be a sense of fullness or thickening.

In the acute episode of recurrent pelvic inflammatory disease, a history of repeated similar attacks of pain, usually in both lower quadrants, with some fever and malaise, may help one to arrive at a correct diagnosis. There may be a history of sexual excesses or physical exertion. Often masses can be felt—which may be unilateral or bilateral—or the adnexal areas are indurated, thickened, and tender. If there is no generalized peritonitis, the patient can move about with little discomfort. An acute appendix in the pelvis or hanging over the brim of the pelvis must be differentiated here.

Currently, the treatment of acute salpingitis is medical. With the use of antibiotics, acute pelvic inflammatory disease may no longer cause occlusion of the tubes with its resultant sterility, provided the treatment is begun early and is adequate. If discovery of acute salpingitis is made at laparotomy, the tubes should not be removed.

Twisted Ovarian Cyst or Twisted Pedunculated Fibroid

The spontaneous torsion of the pedicle of an ovarian cyst is not as frequent a cause of the acute abdomen as some of the other gynecological conditions. It occurs usually in the second to the fifth decades. The ovarian cysts are usually medium to moderate in size with long thin pedicles. The onset is quite sudden (yet a slow twist can produce slow and intermittent pain; it is here that one must rule out tubal abortion) with sharp crampy pains in the lower abdomen, followed by nausea and vomiting. About 85% will reveal a palpable tumor on bimanual examination. Treatment is surgical removal of the affected ovary. Care must be taken to avoid untwisting the pedicle at operation. This is a dangerous procedure for it may release thrombi.

Threatened Abortion

This condition may cause subacute abdominal symptoms and may even suggest an ectopic pregnancy with possible intra-abdominal hemorrhage. Observation is important until either the symptoms subside or the *crampy* pains increase in severity with increased external bleeding and abortion. One must remember that the biologic tests for pregnancy are only positive and helpful so long as chorionic villi are still alive.

Peritonitis may follow a septic abortion. The history of uterine instrumentation is important here. Vaginal bleeding is often present. Pain is usually suprapubic but may radiate to both lower quadrants. The patient is toxic and fever, chills, and sepsis are observed. Pelvic peritonitis is usually present. The modern treatment is conservative, using blood, antibiotics, *etc.* Currettement is rarely done, and only if the degree of bleeding is life-threatening. The history of self-instrumentation of the uterus, with the intent of producing a criminal abortion by the use of various gadgets such as crochet needles, knitting needles, *etc.* may be valuable in arriving at a diagnosis.

Hemorrhage Into a Cyst or Fibroid

Hemorrhage into an ovarian cyst may bring on acute abdominal pain, nausea and vomiting, and even a semi-shock-like syndrome. The bleeding may be intracystic and may become encapsulated as a hematocele which may, of course, be absorbed in time. Occasionally, the cyst may stretch and burst, allowing blood to enter the peritoneal cavity, causing

symptoms of peritoneal irritation.

Sudden hemorrhage can also occur into a fibroid. Acute pain is the result and may be quite sudden and severe.

Sudden hemorrhage into an ovarian cyst may be difficult to differentiate from an ectopic pregnancy. A flat x-ray plate of the abdomen may aid in revealing a dermoid cyst with its ectodermal structures, showing teeth or bone, or showing a "halo" sign due to the sebaceous lipoid-like content. If the encysted structure be that of an endometrioma involving the ovary, the patient will also, as a rule, present the general syndrome of endometriosis — namely, acquired progressive dysmenorrhea, possible sterility, adnexal thickening and fixation, and possible nodular induration of the rectovaginal and posterior cul-de-sac area.

The ovary containing the acute intracystic (intralocular) or subcapsular hemorrhage will reveal, on vaginal examination in most cases, a rather soft, tender, palpable adnexal mass, often previously identified as an asymptomatic ovarian cyst. If a nodular, otherwise firm, fibroid uterus has an area of softtening, it is likely due to red degeneration of one of the fibroids. Still one must keep in mind softening (even in a fibroid uterus) due to early pregnancy.

The acute symptoms may in many cases subside gradually. However, if they persist or get progressively worse, surgical intervention is indicated. The sacculated, hemorrhagic, cystic region can be identified easily and is removed as part of the entire ovarian cystic structure. In the young individual, conservative resection of the ovary must be kept in mind. The treatment of hemorrhage into a fibroid is myomectomy or, in some cases, hysterectomy.

Imperforate Hymen with Hematocolpos, Hematometra, and Possible Hematosalpinx

This condition is one that is often not diagnosed until the patient comes in with acute or subacute abdominal distress. The onset is usually gradual until it reaches the point of acute pain. There may be a history of previous abdominal discomfort, even pain and bloating. These patients are usually past the age of the onset of puberty and may have even reached their eighteenth or nineteenth birthday. They have "not yet started to menstruate." On careful questioning one might get the story that there were somewhat cyclic pains in the lower abdomen with a sense of fullness.

The diagnosis of imperforate hymen is usually made by inspection. An enlarged lower abdomen, with a mass rising above the symphysis pubis (uterus), and a bulging, imperforate hymen, usually of dark blue color, will be noted. The urinary bladder must be catheterized. On rectal examination the markedly distended vagina is felt. Release of this old, "black" vaginal blood is accompanied by a very foul odor.

The treatment consists of crucial incision of the hymen under general anesthesia, with gradual release of the decomposed, tarry blood.

Acute Abdomen in Pregnancy

Pregnancy should not deter one from thinking of *acute appendicitis*. Once acute appendicitis is diagnosed, the management is surgical — the same as it would be in the non-pregnant state. Use of the gridiron incision does not produce any difficulty. The expectant method of treatment of an early case of appendicitis in a pregnant woman is not justified, because true appendicitis is a progressive, unrelenting disease and will terminate in perforation. The anatomic site of the appendix does not alter much due to the growing uterus. At times, diagnosis in the later stages of pregnancy demands the greatest skill. Delay may lead to abscess and possible spreading peritonitis. The large uterus found in the last trimester may indeed become irritable and give rise to painful contractions with abruptio-like symptoms, if a juxta-appendiceal abscess is present. The *modus operandi* is not well known, but reflex contractions due to uterine serositis may be the answer.

Hyperemesis gravidarum may be so severe as to give rise to the question of intestinal obstruction. To differentiate these two conditions, the following points are of assistance. There is no pain preceding the vomiting in hyperemesis gra-

(Continued on page 121)

IV. THE ACUTE ABDOMEN
The Radiological Aspect

JULIAN ARENDT, M.D.*

Surprisingly, many cases of "Acute Abdomen" do not have a suggestive history or localizing physical findings. These obscure cases, with their wide range of diagnostic possibilities, require a complete emergency work-up, including an x-ray examination of the abdomen *and* chest. The roentgenologist who has spent many years in a general hospital and has handled emergency work will remember the many exceptions to the simplifications which teaching and textbook writing demand. He will remember the case of pulmonary thrombosis which came to the hospital with excruciating abdominal pain, the case of massive fecal impaction which almost came to surgery as an acute obstruction, the dissecting aneurysm which imitated a viscus perforation, the pneumonia which had as its only symptom tenderness in the umbilical region, and the volvulus of the cecum which imitated an appendiceal abscess. He will have a small collection of cases which at one time showed irregular gas distribution in the small and large bowel, cases which were too obscure for roentgenological or clinical diagnosis but which cleared up without active medical or surgical intervention.

In all cases of obscure abdominal pain, a chest film as well as a "scout film" of the abdomen should be ordered. These films, whenever possible, should be taken in the x-ray department using all auxiliary devices; improvisation at the bedside, with a portable unit, is hardly worth the effort. The patient should not be prepared for such a scout film — no enema or sedation should be given. However, catheterization of the bladder is indicated.

If technically correct, such a scout film of the abdomen will reveal, almost at a glance, calculi of the gall bladder, kidney, ureter, and pancreas, appendiceal concretions, aneurysms of the abdominal aorta or one of its branches, an abscess or compression of the spine, a perinephritic abscess which obscures the kidney outline and the psoas muscle, an acute dilatation of the stomach, free gas and fluid in the abdomen, and pelvic and abdominal masses — provided there are differences in density setting them off from the surrounding tissues. There might be a suggestion of an intussusception or of a torsion and volvulus which may be confirmed later by barium enema.

All these findings demand clinical evaluation and correlation. The obvious gallstone might not be the source of the symptoms; the gas distention of the bowel loops might be a simple reflex ileus. It is well recognized that the most frequent abdominal emergency, the acute appendix, frequently fails to show definite roentgenological signs unless peritoneal irritation or abscess formation is present. Even perforations of the stomach are known to occur without the clinical signs of prostration and shock and without the roentgenological evidence of a pneumoperitoneum, however small and hidden.

The diagnosis which confronts the roentgenologist most frequently is that of an *ileus*, either of the *obstructive* type or of the *adynamic* type. He bases his diagnosis on the presence of gas and fluid in bowel loops and on the particular arrangement of these loops. It is not rare to find gas within bowel loops. Meteorism is a common finding in cardiacs with sluggish circulation (Figure 1). Patients resting in bed for some time, particularly when immobilized by a cast or when under sedation, develop meteorism. In Hirschsprung's disease, the bowels are markedly distended by gas (Figure 2). Coma and uremia also cause dilatation of bowel loops.

However, the gas in an acute intestinal obstruction of the small bowel (Figures 3a, 3b, and 4), where normally no gas is found, assumes, in a few hours, the char-

* Associate Professor of Radiology, The Chicago Medical School; Director of X-Ray Department, The Mount Sinai Hospital of Chicago.

X-ray photographs included were provided by the Department of Pathology of The Mount Sinai Hospital of Chicago.

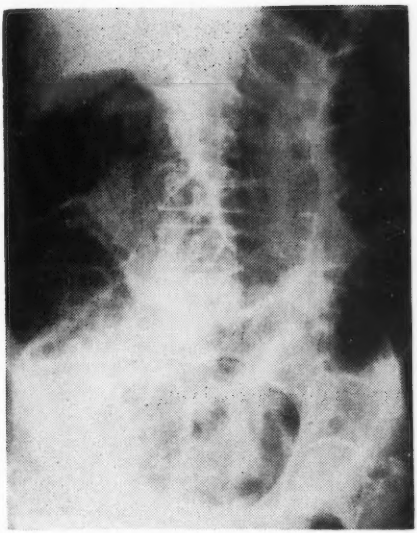

Figure 1

Generalized small and large bowel distention.

M.L., 74 yr. male, one week history of vomiting, cramping pain in abdomen, and "swelling of abdomen."

Recent myocardial infarct; no abdominal pathology.

acter of a forceful dilatation which stretches and accentuates the valvulae conniventes (or Kerkring folds) and gives rise to the typical "herring-bone design." The loops rear over the site of the obstruction and form a stepladder or coiled spring design, while the site of the obstruction remains dense due to fluid accumulation. Following the stepladder down to the area of fluid density we arrive at the point of obstruction. There should be no gas beyond the area of obstruction — if it is a complete one.

At approximately the same time, four hours after the onset of the ileus, fluid begins to accumulate. The source of the fluid is ingested food and liquid and the secretion of glands, which is very likely increased in intestinal obstruction. The demonstration of fluid levels in the small bowel indicates stasis. In intestinal obstruction, the gas chambers and fluid levels frequently combine to form a picture of multiple cascades, arranged at different levels according to the anchorage of the root of the mesentery, which ascends from the right iliac fossa to the upper pole of the left kidney. On fluoroscopy, one can see the fluid levels rise and fall with the peristaltic effort to overcome the mechanical obstruction.

In contrast, the paralytic or adynamic ileus shows no such distinct pattern. More frequently we find here segmental dilatation of small and large bowel loops with wide horizontal fluid levels which can be passively reshuffled by positioning. On the lateral decubitus views, one sees one or more long horizontal surfaces. The rectum and the stomach are usually as distended as the small and large bowel loops.

The paralytic ileus is not always a true paralysis as the intestine can be made to contract by various stimuli. More frequently, the paralysis is due to stimulation of the sympathetic nervous system with its inhibitory effect (Ochsner, Wangensteen). In an acute gall bladder

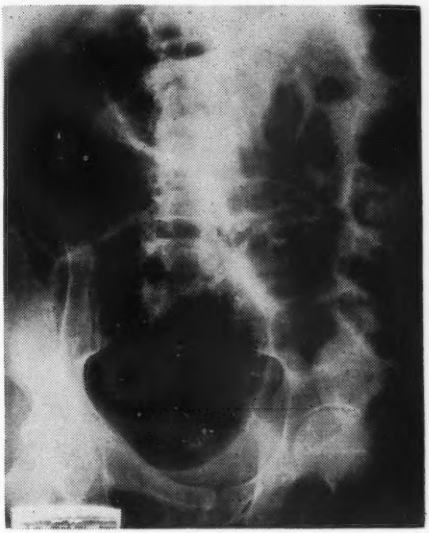

Figure 2

Maximal large and small bowel distention.

S.M., 68 yr. female, history of intermittent abdominal distention and inability to move bowels. No previous surgery.

Megacolon (Hirschsprung's disease).

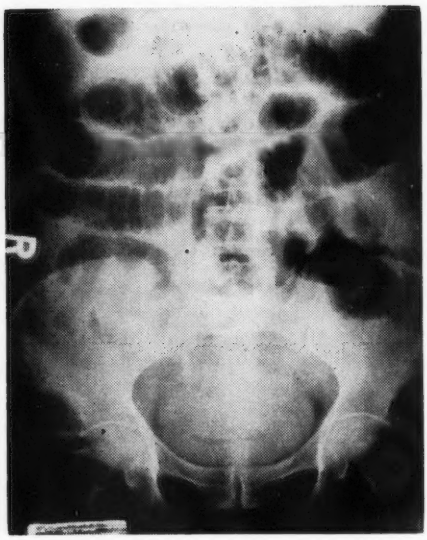

Figure 3a

Small bowel obstruction. Herring-bone pattern. Stepladder leading to density above pelvic inlet.

Z.M., 68 yr. female, colicky pain, vomiting, constipation.

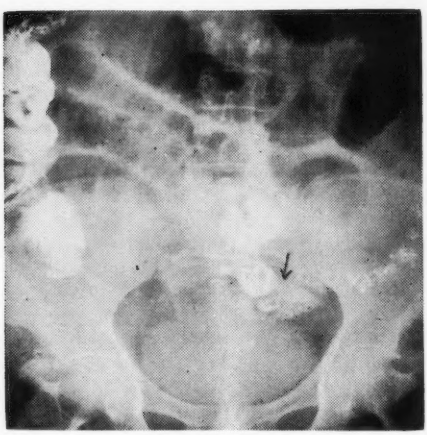

Figure 3b

Same case. Barium enema demonstrates perforation of a sigmoid diverticulum into small bowel loop. (Arrow)

attack as well as in appendicitis and intra-abdominal abscess, the inhibitory effect may be localized to cause a single loop distention, the so-called "sentinel loop." In renal colic and ureteral stone attack, localized bowel spasm and reflex ileus are frequently roentgenological companion signs (Figure 5). It is important in this connection to remember a fact which is frequently forgotten: in 19% of Arneson's cases of renal stone colic no red blood cells were found, and in 37% the chemical test for blood was negative. An intravenous pyelogram, after the acute phase has subsided, is frequently necessary to demonstrate the underlying kidney pathology.

The division between obstructive and adynamic ileus is not too distinct since *combined ileus* occurs, particularly in abdominal inflammation. An appendiceal abscess might cause obstruction as well as inhibition and in such cases of combined ileus it is not unusual to find gas and fluid in the cecum and colon as well as in the small bowel.

Strangulation will modify the picture of an intestinal obstruction. Based on animal experiments, Friman-Dahl has demonstrated that there is scanty gas

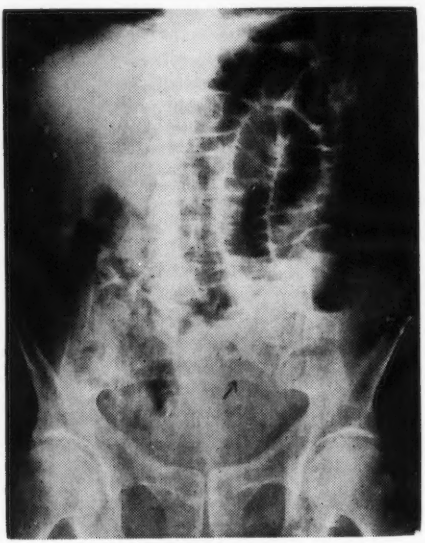

Figure 4

Upper loops are air-distended. Coil-spring pattern. Arrow points to fluid-filled loop.

Dynamic ileus. Small bowel obstruction due to internal hernia of proximal ileum.

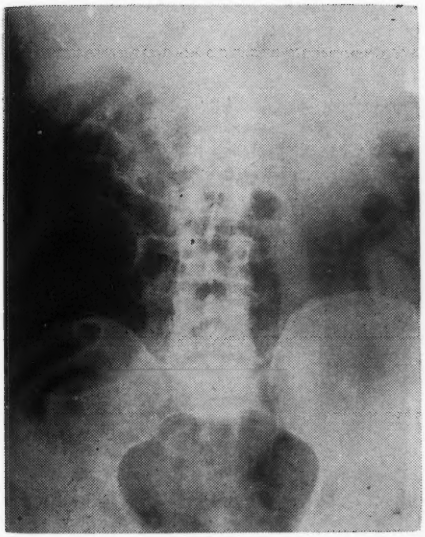

Figure 5

Unilateral small bowel distention.

B.S., 46 yr. female, symptoms and signs of renal colic, history of previous renal calculi.

Reflex ileus accompanying acute renal colic.

accumulation in the pre-stenotic loops, while there is usually a tumor-like density noted in the incarcerated area — consisting of fluid-filled bowel loops. Those fluid levels which are visible are short and scanty. Our own observations have confirmed the author's conclusion that the most severe cases of obstruction frequently show scanty roentgenological findings. The use of the Miller-Abbott tube in such cases is decidedly contraindicated. A thin barium mixture given orally is harmless and might contribute to the diagnosis, if a waiting period of 3-4 hours can be justified.

Among the *circulatory* changes leading to acute abdominal pain of the severest type is *mesenteric arterial occlusion* — most frequently occlusion of the superior mesenteric artery. The physical examination is usually not of much help, but the passage of blood is highly suggestive. The x-ray findings, (Figure 6) which serve as no more than supportive evidence, consist of fluid levels formed not by transudate, but by blood and small and large bowel distention frequently ending abruptly in the region of the splenic flexure. A barium enema, as suggested by Rendich and Harrington, will actually show changed tonus in this region, which I believe is somewhat variable as it denotes Cannon's point (the demarcation of the proximal nerve and blood supply). As the extent of involvement depends on the location of the thrombus, no definite gas pattern can be expected in every case.

In the diagnosis of *local inflammation* of the peritoneal cavity, the observation of the flank stripe and of the properitoneal fat line is of importance. Normally, we find along the flanks, on suitable films taken in the supine and lateral decubitus positions, one or more translucent zones caused by the fatty layers between the abdominal muscles and by

Figure 6

Fluid in lower abdomen with small and large bowel distention limited to ascending and first part of transverse colon.

19 yr. primipara, 24 hours post partum, acute abdominal pain without distention and without rectal discharge.

Small bowel gangrene due to mesenteric artery thrombosis. (Courtesy Dr. D. A. Peckler)

the retroperitoneal fat. In the presence of an abscess which reaches the flank, these clear lines caused by fat disappear or are blurred (Figure 7). If only free fluid is present, it will not obscure the fat line but surround and cap the bowel loops. If the patient is tilted upward, the fluid will leave its flank location and accumulate in the pelvis giving a "new moon" to "full moon" shadow, depending on the amount of fluid.

In generalized peritonitis, however, the flank stripe is frequently not clear but blurred, the subcutaneous tissue is slightly edematous, and the bowel walls and bowel loops are distended.

The diagnosis and identification of *large bowel* obstruction does not, as a rule, present equal difficulties. The colon is more easily identified by its haustral markings, and a contrast enema will usually give us a clear view of such lesions as malignancies and intussuscep-

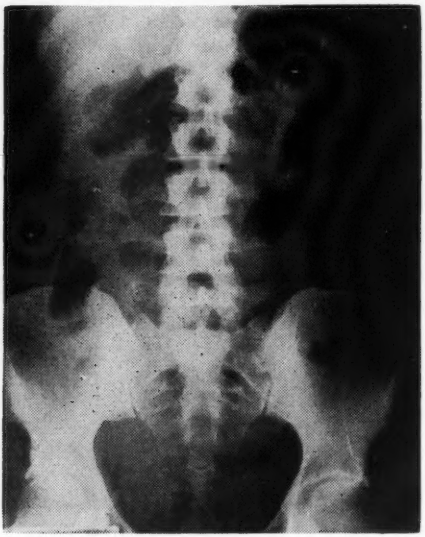

Figure 7

Non-characteristic bowel distention. Arrows point to flank stripe obscured by localized peritonitis.

T.P., 41 yr. male, two day history of sudden, sharp abdominal pain followed by nausea and vomiting.

Ruptured appendiceal abscess with pus and fecal material in abdominal cavity.

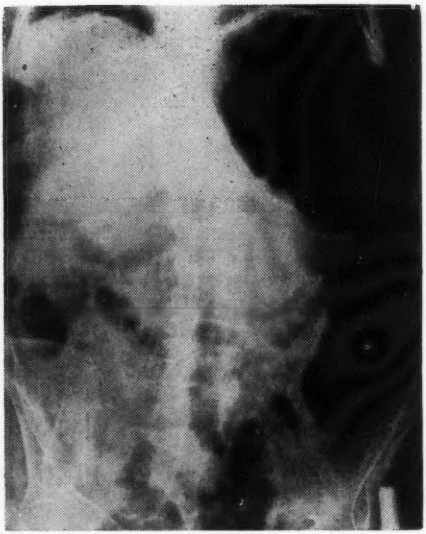

Figure 8

Note abrupt cone-shaped limitation of air distention to the descending colon.

L.K., 75 yr. male, symptoms and signs of "acute abdomen" and extreme distention.

Volvulus of the sigmoid.

tion. A marked cecal distention is characteristic of a closed loop obstruction or of a volvulus of the cecum. A volvulus of the sigmoid occurs frequently with a redundant colon. The acute attack is often preceded by periods of abdominal pain. On x-ray films, (Figure 8) we find a tremendous distention of the twisted loop — which may fill the entire abdomen. Fluid levels are usually absent and there is no gas in the rectum. Barium enema demonstrates the actual area of obstruction, which has, in the case of a volvulus, the appearance of a twisted towel with a corkscrew mucosal pattern. The early diagnosis, which can be materially aided by the x-ray film, is essential to save life; in the past mortality was as high as 40%. We have in our collection several such cases of volvulus of the cecum and of the sigmoid as well as one of the stomach with an axis rotation of 180 degrees, which brought the pylorus into a location above and anterior to the cardia. This latter case responded excep-

tionally well to medical decompression. However, surgery is usually indicated in all cases of volvulus.

It is not and cannot be our intention to give here an exhaustive description of all the roentgenological findings which can be encountered in an acute abdomen. Only a few principles of differentiation have been described. They show, I believe, the advancement made during recent years through attention to detail, refinement of technique, and meticulous observation. An "acute abdomen" was once sufficient diagnosis for the surgeon to open the abdomen and look around. The mortality was high. The improved results of our best clinics are the outcome of early and more specific diagnosis, efficiency of decompression through intubation, and correction of electrolytic imbalance.

REFERENCES

1. Arnesen, A.: The acute kidney stone attack. Z. Urol. Clinic., 45:94, 1939.
2. Frimen-Dahl, J.: Roentgen examinations in acute abdominal diseases, Charles P. Thomas Publ., Springfield, Ohio.
3. Frimen-Dahl, J.: Roentgenoical examinations of ileus, Acta Radiol., 28:331, 1947.
4. Harrington, D.: Mesenteric Thrombosis, Am. J. Roentgenol., 58:637, 1947.
5. Laurell, H.: Acta Radiol., 16:424, 1935.
6. Rendich, R. and Abrams, H.: Ann. of Surg., 102:1040, 1935.
7. Wangensteen, O. H.: Intestinal Obstruction, Charles C. Thomas Publ., Springfield, Ohio.
8. Wangensteen, O. H.: Radiology, 35:680, 1940.

Symposium:
A Gynecologist's Approach—

(Continued from page 115)

vidarum, while the pain of intestinal obstruction is griping and intermittent and usually precedes vomiting. In hyperemesis, any pain that does occur is due to straining and is in the abdominal wall. Food, especially glucose, will temporarily relieve the symptoms. In intestinal obstruction the intake of food or drink starts a peristaltic wave, which initiates more pain and vomiting. If the vomiting is part of a toxemia, a catheterized urine specimen will usually show albumin, especially if the toxemia is severe.

In *pyelitis of pregnancy* the pain is found more on the right side, in the loin area, and tends to radiate along the course of the ureter to the bladder region. If very acute and severe, it may be associated with vomiting. The onset is usually more sudden than that of acute appendicitis. Often palpation of the kidney on that side reveals tenderness. The temperature is higher than in acute appendicitis, being about 100°F. or over, with a relatively slower pulse. Chills may occur (practically never in appendicitis). The urine is usually cloudy and shows pus and organisms. (The urine may be clear if the affected ureter is blocked.) Treatment is symptomatic, along with the use of antibiotics.

Other genito-urinary conditions in pregnancy which may simulate an acute abdomen and are worthy of mention are Dietl's crisis due to kinking of the ureter (more common in multipara), nephrolithiasis, and acute urinary retention giving rise to a large palpable bladder.

V. CLINICOPATHOLOGIC CONFERENCE

Presented at Mount Sinai Hospital, Chicago, Illinois

DR. L. FELDMAN, Chairman DR. I. DAVIDSOHN, Secretary

Abstracted by DR. ISADORE KLEIN

First Admission: The patient, a forty year old white female, was admitted to The Mount Sinai Hospital on May 4, 1946. At that time she was near term in her fourth pregnancy. She was in false labor and was discharged on the same day.

Second and Third Admissions: On both May 14 and June 8, 1946 the patient was readmitted to this hospital, but again was found to be in false labor. Discharge from hospital occurred on the following day in each instance.

Fourth Admission: The patient was admitted on June 12, 1946, in labor, and this time delivered a normal white male child. The postpartum stay in the hospital was without incidence and she was discharged on June 19, 1946.

Fifth Admission: The patient was readmitted to The Mount Sinai Hospital on February 28, 1947 with complaints of hypermenorrhea and metrorrhagia since November of 1946.

Physical examination at this time was essentially normal. The abdomen was quite obese and revealed an old, well-healed, midline scar. No masses were present, no tenderness was elicited, and no distention was noted. The liver, kidneys, and spleen were not palpable. There was no costovertebral tenderness. The abdominal scar was due to an appendectomy performed in 1925 and an uterine suspension in 1927. The patient was discharged on March 4, 1947.

Sixth Admission: The patient was readmitted to The Mount Sinai Hospital on October 27, 1947 with the complaint of a sudden onset of left lower quadrant pain three and one-half days prior to admission. The pain was described as constant with intermittent cramp-like seizures. Treatment, consisting of hot baths and the application of heat to the left lower quadrant, seemed to improve her condition. She was discharged on October 29, 1947, much improved.

Seventh (Final) Admission: The patient was readmitted to this hospital on October 9, 1952, with the complaint of severe pain in the right upper quadrant. She was now forty-three years old and had experienced belching and postcibal epigastric distress for the past two years. In 1950, roentgenologic studies had revealed a gallbladder containing negative shadow stones. Two days prior to this hospital admission the patient's first attack of severe epigastric pain occurred. This pain radiated to the subcostal and dorsal regions, and was accompanied by nausea, vomiting, and eructation. These symptoms persisted until the time of admission. At no time during these attacks did the patient notice any dark urine. There was no past history of jaundice, chills, or fever.

Physical examination revealed a well developed, well nourished, markedly obese white female. The blood pressure was 112/88. The heart and lungs were essentially normal. Abdominal examination revealed no masses and the liver, kidneys, and spleen were not palpable. There was tenderness and rebound tenderness in the right upper quadrant and in the lower abdomen. There was no rigidity of the abdomen. The impression at this time was that of an acute exacerbation of a chronic cholelithiasis.

On October 21, the patient was still complaining of pain, although it was not as severe as before. The temperature had risen to 100° F. A Levin tube was inserted and was draining well. The urine examination revealed a 1% sugar. The blood sugar level was 154 mg.%. The patient was considered a diabetic despite the absence of diabetic symptomatology. The patient's symptoms continued unchanged and on October 28, 1952, an operation was performed, at which time a small, thick, inflamed, contracted gall-

bladder was found wedged into the liver. There was a fistulous connection between the duodenum and the fundus of the gallbladder. The common duct was described as being free of stones. A cholecystectomy and a transverse closure of the duodenal fistula were performed. The wound healed without incidence. However, the patient began to have progressively more severe episodes of vomiting post-operatively, and on November 9, 1952, it was noticed that tremendous quantities of vomiting had occurred on the previous evening. The patient was described as cold and in a shock-like condition on the morning of November 9. In addition, there was a tremendous distention of the left upper quadrant. The distention was relieved by the passage of a Levin tube, through which a large volume of yellow-tinged fluid was removed from the stomach. Bowel sounds were present. The patient was afebrile.

Rectal examination at this time was essentially negative. A small amount of stool was present. The impression at this time was that of an acute gastric dilatation. To be ruled out was a postoperative mechanical obstruction of the duodenum due to closure of the cholecystoduodenal fistula. Later that afternoon, at 4:20 P.M., the patient was found in shock and no blood pressure was obtainable. There was no pulse and the skin was cold and clammy. The abdomen was extremely distended throughout, with only occasional peristaltic sounds audible. The abdomen was soft. Intravenous fluids were started and 20 mg. of Desoxyn was given. At 4:50 P.M. the blood pressure was 75/60 and the pulse was palpable and regular at a rate of 140 beats per minute. No urinary output was reported during the day. At 5:30 P.M. the pulse rate was still 140 per minute but the systolic pressure had fallen to 60 and the diastolic pressure could not be ascertained. The abdomen remained soft. The patient was complaining of pain. A catheter was inserted and two ounces of concentrated urine was withdrawn.

The impression at this time was that of hypochloremic alkalosis with shock and renal shut-down. Therapy consisted of 2000 cc. of 5% glucose in saline plus 60 mEq. of potassium chloride, 10 mg. of calcium gluconate, and 1000 cc. of 5% glucose in water with 40 mEq. of potassium chloride. At 7:00 P.M. the blood pressure had risen to 132/110.

The patient's course, in spite of this treatment, was progressively downhill and she expired at 12:35 A.M. on November 10, 1952.

Discussion

Dr. L. Feldman: As one studies the urinary findings in this patient, the presence of glucosuria is interesting to note. During the last admission, the white blood count was also very interesting, because the patient came in with a leukocytosis of 11,000 with 50 per cent segmented cells. Later, there were 33 per cent segmented cells and the white count came down to 9,800 and then to 8,800. November 9, 1952, after the operation, the white count again rose to 23,400. There was no shift to the left in spite of the increase in the white count during this period of complication. (See Laboratory Data.)

Dr. W. Smallberg: In the clinical abstract it is stated: "The abdomen was extremely distended throughout, with only occasional peristaltic sounds audible." Then there is a sentence, "The abdomen was soft." To my knowledge, the abdomen of this patient was always soft, except earlier that day when she had an acute dilatation. This was a confusing aspect. There were bowel sounds at all times, and after her operation she continued to have bowel movements. Since I am familiar with the autopsy findings, I would rather not enter the discussion until after the autopsy findings are given.

Dr. W. W. Masur: In going through the record, it seems to me that this patient had a chronic cholecystitis with cholelithiasis, with repeated exacerbations. It is stated that on the sixth admission she had experienced pains in the left lower quadrant which had persisted for three and one-half days. The question arises whether at that time she had a perforated gallbladder with formation of a cholecystoduodenal fistula. Nothing is mentioned here regarding the treatment that was instituted, except the use of

LABORATORY DATA

Blood Counts:	RBC (mill.)	Hb. (Gm.)	C.I.	WBC	Differential Count (in %) Segs	Eos	Lymphs	Metas	Stabs	Monos
5th admission										
2/28/47	3.71	12.3	0.99	9,200						
6th admission										
10/2/47	4.34	14.0		10,750	70	1	29			
7th admission										
10/19/52	5.28	16.9	1.01	11,400	50	1	14	1	14	10
10/21/52	5.17	15.6	0.96	9,800	33	1	22		24	19
10/22/52				8,800	23	2	37		19	18
10/23/52				11,450	16	2	34	2	38	8
10/24/52				16,300	58		26		12	4
10/25/52	4.60	14.8	1.02	13,900	24	3	24	2	34	13
10/26/52				14,750	66		20		6	8
10/27/52				12,450	30	1	34		26	8
10/31/52	3.63	11.1	0.96	13,000	68	1	24		1	6
11/3/52	4.75	14.4	0.97	16,400	57		16	3	16	8
11/9/52	5.60	16.5	0.94	23,400	59	1	15	4	16	5

Some atypical monocytes, atypical lymphocytes, and toxic granules were reported on several occasions. On 10/24/52 some plasma cells were reported.

Urinalyses:	Color	pH	Sp.Gr.	Prot.	Sugar	Acet.	Casts	RBC	WBC	Blood
7th admission										
10/20/52	Amb.cldy.	5.0	1.021	1+	1.0%	0	0-1	1-2	2-6	
10/27/52	Dark amb. cloudy	6.0	1.017	Tr.	1.0%	0	Occ.	1-3	3-15	Ft. Tr.
10/28/52	Amb.cldy.	6.0	1.009	Tr.	0.2%	0	0	5-10	8-10	Ft. Tr.
10/29/52	Amb.cldy.	6.0	1.024	Tr.	0	0	0-2	7-8	80-100	Ft. Tr.
11/4/52	Dark amb. cloudy	5.0	1.025	Tr.	0.2%	0	0	3-4	25-30	0

Blood chemistries:	Sugar	UreaN	Creat.	Chlorides	Amylase	Potassium	CO_2
7th admission							
10/20/52	154	42.9	1.9	88.2 mEq/L			
10/21/52					26 mg/100 ml	4.3 mEq/L	
10/27/52		18.0		77 mEq/L			29.3 mM/L (65 vol%)

Serologic test for syphilis: 10/28/47 - Kolmer, Kline, Kahn: Negative.

Prothrombin time: 10/24/52 - 14.6% (clotting activity 85% of normal).

Test for infectious mononucleosis: 10/23/52 - Presumptive test: 1:7 (positive).
Heterophilic antibody test: Negative.

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heat. She was discharged two days after admission. Another point worth discussing is that of the signs of diabetes mellitus during the last admission. The question that should be raised here is that of the possibility of a direct extension of the inflammatory process into the pancreas as a consequence of the cholecystoduodenal fistula with the development of a concommitant pancreatitis causing a glycosuria and an elevation of the blood sugar. Also, I would like to know if there were stones in the gallbladder at the time of surgery. If not, we have to assume that the stones passed through the fistula.

Dr. L. Feldman: The patient had cholecystic disease, glycosuria, and hyperglycemia. There is no history of any previous glycosuria. Therefore, let us consider whether or not the pancreas could be involved.

Dr. H. Rony: There is a possibility that hyperglycemia and glycosuria might be a sign of acute or subacute pancreatitis. It might help to know if this patient had a family history of diabetes. There was no note on the chart about that. Otherwise, it would be difficult to decide if this was an incidental appearance of diabetes, which is, after all, possible, or if it was due to secondary pancreatic involvement. In cases of direct involvement of the pancreas causing hyperglycemia and glycosuria, one would expect the condition to be severe, although I suppose milder degrees of pancreatic disease could present findings such as these.

I would like to say just a word about the hypochloremic alkalosis. That, of course, can be explained on the basis of the severe vomiting that this patient had. With the loss of excessive quantities of hydrochloric acid, hypochloremia and hypochloremic alkalosis will result. Apparently that was the reason in this case. However, the alkalosis was properly treated and apparently was not the cause of death.

Dr. L. Feldman: What was the gross appearance of the pancreas when you removed the gallbladder? I would also like to ask Dr. Smallberg about the possibility of having narrowed the canal producing an obstruction by virtue of the transverse closure.

Dr. W. Smallberg: The transverse closure was performed in order to prevent narrowing of the duodenum at the point where it had been opened longitudinally.

Dr. L. Kolb: There are two points here that I would like to discuss. One is the x-ray examination in 1950 which showed a gallbladder containing negative shadow stones. Obviously this woman had bilirubin and cholesterol stones extending back to at least 1950. The question arises as to why her gallbladder was not removed in 1950 when the diagnosis was made in view of the fact that stones of this kind usually cause trouble. This is an example of the difficulties that may arise from not removing gallbladders with stones. The second point is: this patient was admitted on October 19, 1952 with acute cholecystitis, and was not operated upon until October 28, 1952. In cases of acute cholecystitis, most men advise that the operation be done in the first 48 hours. If not, the gallbladder should be allowed to cool off for six weeks before surgery. I do not know the cause of death, but there is a possibility that with the gastrectasis, although very late, there was a slow leak in the duodenum due to the tremendous pressure created by the overdistention of the stomach. As far as the fistula is concerned, someone previously suggested that the pain in the left lower quadrant in October, 1947 had been due to cholecystitis with perforation. One could then assume that the fistula had been formed at that time. Had that occurred, I doubt that stones would have been seen in 1950 because they would have passed out through the fistula. Although it was not mentioned, I assume that no stones were found in the gallbladder at the time of operation. They probably had passed into the bowel.

Dr. L. Feldman: Summarizing at this point, we have a medical man who suspects acute pancreatitis and a surgeon who has considered a ruptured viscus. A rupture either at the site of operation or in an acute peptic ulcer could have been aided by the distention which this patient had postoperatively. It seems to me that the rupture of the viscus is a very likely possibility.

Dr. H. F. Weisberg: First, I would like to disagree with the evidence that this is a case of diabetes. The history is not complete. This patient received two liters of glucose solution on the night of admission—the blood was drawn the following morning. The finding of 1% sugar in the urine and a blood sugar level of 154 mg % without a suggestive history or other symptomatology does not warrant this diagnosis. We should at least have more blood sugars and a further work-up. The criteria of the American Diabetes Association for the diagnosis of diabetes are quite definite. A fasting blood sugar level of 150 is suspicious if the Folin-Wu method is used. We do the Somogyi blood sugar in which a fasting level of 120 is considered upper limit of normal. We may suspect that

this patient had a renal glycosuria, because ordinarily the "threshhold" is about 180 mg. % rather than 150. I do not think diabetes due to direct extension into the pancreas existed because pancreatitis with a high amylase would be present. In this case, the amylase was 26 mg. %, a value which is far below normal and one which would speak for poor liver function. This fits in with the hypothesis of a renal glycosuria because, with poor liver function, the liver glycogen would be broken down and glycosuria could exist in the face of a low blood sugar level. I therefore do not think that the diagnosis of diabetes is warranted.

However, I do think that we have some questions to ask. This patient was vomiting tremendous quantities of fluid and yet there were no laboratory evaluations from October 27th until her death on November 10th. Thus, for a period of two weeks there was no electrolyte evaluation. It was very admirable to have a hematology check every second day for the white blood count, but this patient was a surgical problem; vomiting with loss of electrolytes and water imbalance. Hypochloremic alkalosis with shock and renal shut-down was suspected. Only 2 ounces of urine were obtained by catheterization. Yet, at the same time, 100mEq. of potassium were given in the course of one day. Such a large quantity of potassium given intravenously in the face of oliguria is sufficient to result in hyperpotassemia, a factor which could have been contributory to the death of this patient.

Dr. L. Feldman: Someone should discuss the possibility of bile peritonitis, which can occur postoperatively. Bile peritonitis can give a high leukocyte count. Therefore, I would like to direct your discussion into that channel: the possibility of bile peritonitis instead of a ruptured viscus. Does anyone care to comment on this?

Dr. W. W. Masur: It seems to be very unlikely that there was a perforation of the duodenum. This complication usually occurs earlier, on the fourth or fifth postoperative day. In addition, the absence of abdominal findings, peritonitis and fever speak against perforation. The only thing that is mentioned is the gastric dilatation. Perforation undoubtedly would give rise to a local infection or even abscess formation. Bile peritonitis gives very violent symptoms, the patients are very sick, usually with a high temperature and findings of abdominal distention.

Autopsy Findings

Dr. I. Davidsohn: The type of lesion represented by this case is not too rare, if one considers that the first case was described in 1654, and that in 1914 a review paper on this condition listed 342 cases reported until that time.

The stomach, as stated in the clinical description, was markedly distended and contained a great deal of fluid, as did the small intestines, presenting the picture of a paralytic ileus. The duodenum showed an opening in the area which had been sutured during surgery. It had opened again, but did not communicate with the peritoneal cavity. Instead, it opened into the fat to which this part of the duodenum had been fastened by the surgeon. There was no peritonitis. The reopening of the suture did not lead to complications. It was through this open-

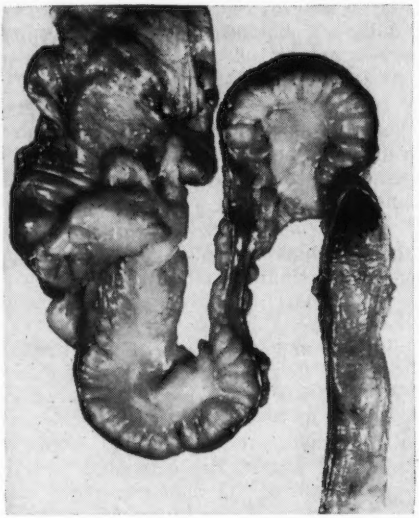


Figure 1
Gallstone obstructing lumen of ileum 45 cm. above ileocecal valve.

Figure 2
Jejunum: Ulcerative pseudomembranous enteritis.

ing that the gallstones originally had passed from the gallbladder into the duodenum. A microscopic section through this area showed reactive changes with remnants of suture material in the wall.

The significant pathology was found 45 cm. proximal to the ileo-cecal valve, in the terminal ileum. Here (Figure 1), a gallstone can be seen obstructing the lumen. The ileum above was dilated; the distal ileum was collapsed. A review of the literature reveals that this condition may be diagnosed with the help of x-ray. Rigler, *et al.*, reported that 13 of 14 cases that came to his attention or that were studied in his hospital were diagnosed with the help of the x-ray.[1]

The gallstone measured 30 mm. in one diameter, and about 25 mm. in the other. It is stated in the literature that this is about the critical size of gallstones responsible for intestinal obstruction. Gallstones of that size have a tendency to be arrested, to produce spasm of the bowel, and to cause an obstruction, usually in the lower ileum. Intestinal obstruction by gallstone is known to have an extremely high mortality—up to 75 per cent, even after the gallstone has been removed. As a result of the extreme distention, the ileum and jejunum showed diffuse ulceration of the mucosa. (Figure 2) Ulcers were present even in the stomach.

You may remember that Dr. Smallberg emphasized that bowel movements were present until the end. It may well be that the dilatation seen at autopsy occurred only during the last day or two, the obstruction being incomplete until then. Ulceration of the mucosa is the usual complication of this type of obstruction. Fluid pours into the lumen; the intestine becomes dilated; there is ischemia of the wall; and eventually ulceration occurs. Microscopic sections of the jejunum show defects in the mucosa. Some defects were deep, reaching almost to the serosa. (Figure 3) This might have led to a rupture, especially in view of the fact that the serosa was diffusely inflamed and infiltrated. The inflammation must have been there for some time for, under high power magnification, one sees necrosis of the mucosa and submucosa, as well as the diffuse inflammatory reaction of the subserosa. In view of these findings, it is remarkable that peristalsis was going on. The anatomic evidence, gross as well as

Figure 3
Jejunum: Acute ulcer destroying mucosa and submucosa. x6

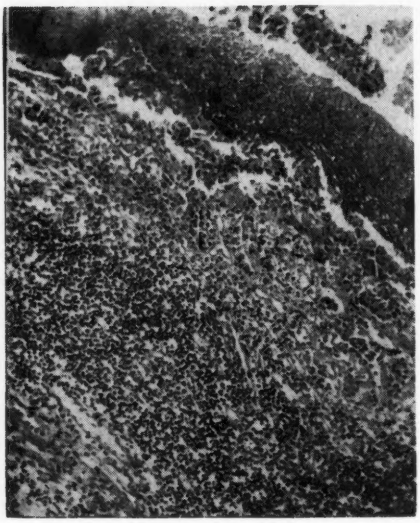


Figure 4
Jejunum: Fibrinous membrane covering ulcer. x110

microscopic, is certainly in accord with a paralytic ileus.

In another slide (Figure 4), can be seen a fibrinous exudate on the surface of the mucosa and inflammation of the remaining layers. Under high power magnification, the membrane covering the mucosa gives the appearance of a so-called pseudodiphtheritic enteritis. In the stomach, there were areas of erosion with superficial necrosis. This change, no doubt, was terminal.

The liver was almost twice its normal size, and showed diffuse degenerative fatty changes. The yellow color was quite pronounced on the fresh specimen. According to the microscopic changes, this liver certainly could not have functioned very efficiently. (Figure 5) The liver parenchyma was replaced by fat and showed far advanced fatty degenerative changes. Changes in the periportal spaces were those of chronic cholangitis, which is frequently an accompaniment of chronic cholecystitis and cholelithiasis. The gallbladder, which was removed surgically, grossly showed advanced chronic inflammatory changes. Although no stones were seen grossly, microscopic sections of the markedly thickened gallbladder wall showed cholesterol deposits. Similar changes were found in the bed of the gallbladder. Some areas also showed deposits of bile. These changes in the wall of the gallbladder tell the story of how this kind of a perforation takes place. First, there are gallstones; then there is an acute ulcerative inflammation. Sometimes this is actually gangrenous. Particles of stones, small stones, or inspissated cholesterol containing biliary matter may become lodged in the wall, forming a calculus or granulomatous cholecystitis, as in this case. Eventually, there is a pericholecystitis, adhesions form between the gallbladder and the duodenum, and the whole wall becomes acutely inflamed. Usually a gangrenous inflammation then develops, resulting in the perforation. In some case reports the authors emphasize that there is an acute subsidence of the pain and disappearance of the physical findings characteristic for empyema of the gallbladder when the perforation takes place and when the stone passes into the duodenum.

Our patient, no doubt, was in shock. We do not need the corroboration of the clinical history, if we look at the kidneys. The kidneys were enlarged and

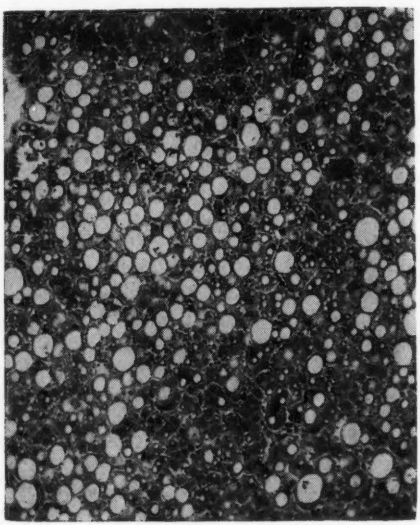


Figure 5
Liver: Diffuse fatty degeneration. x110

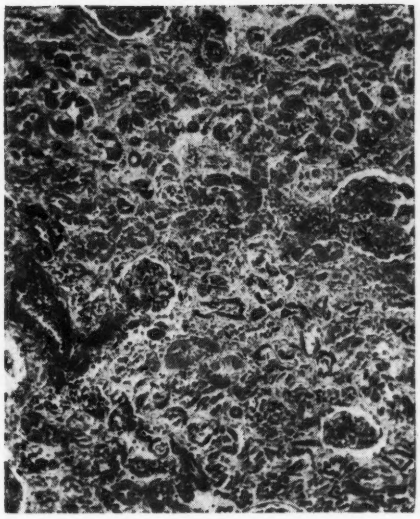


Figure 6
Kidney: Diffuse parenchymatous degeneration. x110

Figure 7
Kidney: Regeneration of tubular epithelium. x440

showed diffuse parenchymatous degeneration, especially of the tubules, with areas of hemorrhage. (Figure 6) The glomeruli were well preserved, but there was advanced necrosis of the tubules in some places and less severe damage in others. In some areas, regeneration of the epithelium was pronounced, as is seen frequently after transient renal ischemia. (Figure 7) Calcium deposits were also seen. The tubular changes were characteristic of what we used to call lower nephron nephrosis.

In summary, this was a case of acute high intestinal obstruction caused by a gallstone which had perforated into the duodenum sometime in the past; resulting in the complications of shock and renal damage.

A review of the literature brings out several points of interest. In 10 per cent of cases with cholecysto-enteric fistulas, an acute intestinal obstruction eventually develops. Statistical reports indicate that about 2 per cent of all cases of acute intestinal obstruction are due to obstructing gallstones. This is not a negligible figure. On the other hand, various accidents influence the incidence of this lesion. For example, one writer reports 10 cases seen in Saint Vincent's Hospital in New York in 13 years—6 of these had occurred in one year. In New York's Bellevue Hospital, a much larger institution with many more cases, not one such case was observed from 1928 to 1948.

Anatomic Diagnoses

GALLBLADDER: Status after cholecystectomy. DUODENUM: Status after repair of choledochoduodenal fistula. SMALL INTESTINE: Obstruction by gallstone; dilatation (paralytic ileus); enteritis, acute, ulcerative and pseudodiphtheritic. STOMACH: Dilatation; erosions, acute, multiple. SPLEEN: Congestion, acute. LIVER: Fatty metamorphosis, extensive; perihepatitis, subacute, focal (gallbladder bed); cholangitis, chronic. KIDNEYS: Nephrosis, acute; lower nephron nephrosis. PANCREAS: Secretory stasis. ABDOMINAL WALL: Surgical incision, recent; umbilical hernia. OVARIES: Fibrosis. UTERUS: Endometrial atrophy. APPENDIX: Status after appendectomy.

Cause of Death: Paralytic ileus due to intestinal obstruction caused by gallstone.

Question: You referred to the location in the lower ileum as typical for obstruction due to gallstones. Why is that so?

Dr. I. Davidsohn: Obstructing gallstones are found most commonly in the lower ileum, sometimes they may be found as low as the ileocecal valve. The lumen of the ileum in that region becomes progressively narrower until a stone about one inch in diameter cannot pass through it. Sometimes the stones are found in other parts of the small intestines, for example, in the duodenum. There is even one case on record where a stone was present in the stomach and caused intermittent obstruction.

Dr. L. Feldman: We have here a group of men who knew that there was a fistula. Yet no one asked about the fate of the gallstone. We cannot really blame the surgeon, because when he is operating he has plenty to do and cannot always think of various eventualities. But, we here, at our ease, should have thought of the migration of the stone into the lower part of the gastrointestinal tract. It shows that it takes a long time to become smart in medicine.

Dr. L. Aries: I had seen this patient for the last four years and, knowing that she had gallstones, had repeatedly asked her to be operated upon. She was raising two small children and never could find time to go to the hospital. When she finally came into the hospital she had acute empyema of the gallbladder with high fever and was extremely sick. We kept her in the hospital for about a week or 10 days before she was ready for surgery. During that time, she developed repeated attacks of colic, which we interpreted as being due to the gallbladder. She developed an acute dilatation of the stomach, which responded very quickly to Levin suction. She continued to have bowel movements. It was the intermittent obstruction by the stone that completely misled us. We were not thinking of the stone at all. She most likely had repeated episodes of incomplete obstruction, which gave the picture that finally precipitated her death. Dr. Smallberg was here with her all day on the Sunday that she died, and the clinical picture was not that of an obstruction, because of the fact that she had bowel movements with enemas, and was passing gas at intervals. The fistulous communication at surgery was about 1 cm. long. A flat plate of the abdomen postoperatively could probably have given us the diagnosis.

Dr. I. Davidsohn: I would like to add to what Dr. Aries has said that one must consider not only the size of the stone, but also the fact that spasm of the bowel wall develops and is responsible for the final catastrophe. Also, these obstructions may be intermittent. Furthermore, when a great deal of fluid accumulates, it may cause strangulation by producing a kink. In other words, to the stone and the spasm is added the accumulation of fluid, which may cause a kink and the final, fatal obstruction.

REFERENCES

1. Rigler, Leo. G., Borman, C. M., and Noble, John F.: Gallstone obstruction; Pathogenesis and roentgen manifestations. J.A.M.A., *117*:1753-1759, (Nov. 22) 1941.

AN ANALYSIS OF THE RANSON PYRIDINE SILVER STAIN

MARVIN W. ROSENZWEIG, A.B., JOHN J. SHEININ, Ph.D., M.D., Sc.D., and GEORGE CLARK, Ph.D.*

One of the most widely used variants of the Cajal type of block silver stain for nerve fibers is that of Ranson[1]. The usefulness of this stain, as well as its limitations, are well attested to by the many variants that have been proposed. With the variations, as with the original descriptions of Cajal and of Ranson, the recommended method has been outlined with little discussion of the various steps which led to the development of the method, and with little or no consideration of the need for exactness of timing or of concentration of the various reagents used. This criticism applies not only to the descriptions of this particular method, but also to most descriptions of staining methods. We have attempted to vary widely each step of the Ranson method in the hope of determining just how critical each step might be, what line of experimentation might lead to the development of a more satisfactory procedure, and finally, to determine if this method might serve as a model for testing other staining procedures. The latter is important in that the lack of detailed information concerning the effect of variations in staining and of slight differences in concentration of staining solutions may render difficult the reading of the prepared sections.

Methods

The test material was dog spinal cord cut into blocks about 3 mm. thick and fixed by immersion for 48 hours in absolute ethyl alcohol containing 1.0 cc. of a concentrated ammonia solution per 100 cc. of alcohol. Each of the remaining steps of the method was varied systematically as shown in Table I. For convenience, those variants of the method with somewhat similar timings were arranged in four groups and separate dogs used for each group. Each set of step variants included one with the recommended time or concentration of which half were prepared from one dog and the remaining half from another. These served as controls for the consistency of the method from animal to animal and from block to block in the same animal.

After staining, the blocks were dehydrated, embedded in paraffin, and sectioned serially at 10 mμ. Every fifth section was mounted. Observations were made on alternate sections throughout each series with various items graded on an arbitrary scale. The following points were considered: differentation of fibers in the outer, middle, and inner thirds of the white matter; differentation of fine fibers and of neurofibrils in the grey matter; and the general contrast in the grey matter. These observations on each series were averaged and are expressed graphically in Figures 1-6.

TABLE I

OUTLINE OF RANSON PYRIDINE SILVER STAIN WITH THE VARIANTS USED.

The recommended time or concentration is underlined.

1. Fixation absolute alcohol and ammonia (1 cc. conc. solution of ammonia in water to 100 cc. of alcohol). 48 hours immersion.
2. Secondary fixation 5% solution of pyridine in distilled water, 0, 6, 12, 12 (p.f. 35) *24*, 36, 48, 60, 60 (p.f. 10) 72 hours.
3. Wash in frequent changes of distilled water. 0, 0 (8 rin), 1, 2, 4, 8, 12, *24*, 48, 96, 192 hours.
4. Stain in a solution of silver nitrate in distilled water.
   A. Variation in staining time. $AgNO_3$ concentration 2%. 1, 4, 12, 32, 48, *72*, 96, 120, 144, 168 hours.
   B. Variation in concentration of staining solution—time 72 hours. 0, 1/4, ½, 1, *2*, 4, 8, 16, 32, 64.
5. Reduce in pyrogallic acid solution in 5% solution of formalin.
   A. Variation in reduction time pyrogallic acid concentration 4%. ½, 1, 2, 4, 8, 16, *24*, 32, 40, 48 hours.
   B. Variation in concentration of pyrogallic acid. Reduction for 24 hours. 0, ½, 1, 2, *4*, 6, 8, 10, 12 grams.

* Department of Anatomy, The Chicago Medical School.

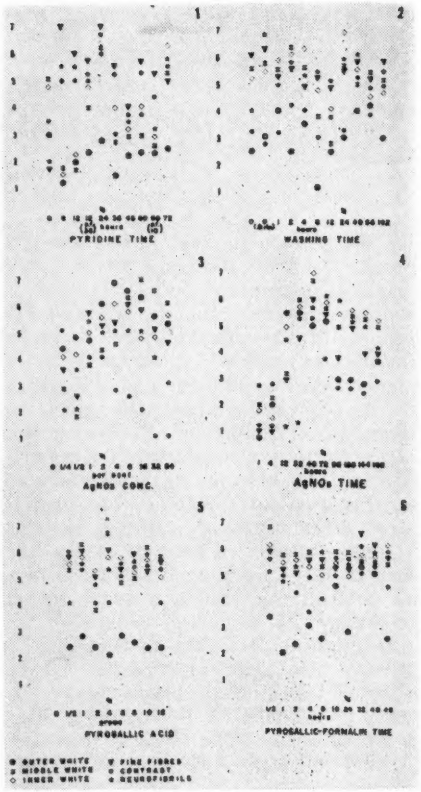
Results

There is some variation in blocks stained by the standard technic as will be seen in Figures 1-6. In each of these, the standard is marked by an arrow. This variation must be considered in evaluating the variant technics. The standards in Figures 1, 5, and 6 were from one dog, and 2, 3, and 4 from another. There is less variation in the first set than in the second, and those of the first set are distinctly better than those of the second. However, all are sufficiently well stained to be quite usable.

It is evident (Figure 1) that the postfixation in aqueous pyridine is an essential step. However, the exact timing is non-critical, except that the postfixation should last at least 12 hours. Washing after postfixation (Figure 2) is also a non-critical step and apparently could be omitted with no deterioration of the quality of the stain. The concentration of silver nitrate in the staining solution is relatively critical. Above 2%, the contrast in the grey matter becomes markedly less, and below 1% the staining is inadequate. However, the impregnation is more complete with the higher percentages and the possibility should be considered that if very thin sections were cut, the higher silver concentrations might prove advantageous.

The length of staining time is also relatively critical. With the shorter staining times, the periphery of the block is not overstained which might prove advantageous in cases where differentation of peripheral structures is important. Considering the entire section, it appears that 32 hours is a minimum and that 72 hours, the standard, is perhaps a maximum.

The concentration of pyrogallic acid is not critical. The 5% formalin is not adequate for proper reduction but all concentrations of pyrogallic acid produced usable slides with no more variation than was seen among the various blocks stained by the standard method. Similarly, the length of the reduction time is not critical and can be varied widely with little change in the finished product.



Discussion

Although these experiments offer no indication as to the direction of experimentation which might lead to the development of a more satisfactory stain, considerable insight was obtained into the actual mechanics of the method. It is quite obvious that much of the variation from block to block, which we have routinely found in using the stain, must be expected to occur regularly, although the causes remain obscure. Equally obvious is the fact that much of the slavish adherence to recommended times and concentrations of reagents was wasted. The washing after postfixation (step 3) might well be omitted. Only with the silver concentration (step 4a) and time in the silver bath (step 4b) is there any indication of narrow limits. Here the range that will give best results seems

to be from 1-2% silver nitrate concentration and from 32-72 hours in staining time. Some years ago, Davenport[2] stated that the pyrogallic acid was the essential element of the reducing solution, and that variations in the amount of formalin were not important, but his series was quite limited.

It is apparent that the analysis of other staining methods by the procedure used in this study might prove quite profitable. The unexpected finding that the washing time after postfixation in pyridine is not critical, suggests that, perhaps, in the Bodian protargol method, increases in washing time might prove advantageous. Similarly with stains of the Mallory and Masson type, a systematic variation of the various steps might yield significant results.

Conclusions

Systematic variation of the various steps of the Ranson pyridine silver stain reveals that in most cases the actual concentrations of reagents and the length of the various steps is relatively uncritical. Only with the strength of the silver impregnation bath and the length of staining must care be used, and even here the range is considerable.

The procedure used could probably profitably be applied to other staining technics.

REFERENCES

1. Ranson, S. W., Non-medullated nerve fibers in the spinal nerves. Am. J. Anat., 12:67-88, 1911.
2. Davenport, H. A., Block staining of nervous tissue with silver. Study of fixatives, lipid solvents and reducing solutions. Stain Techn., 5:139-147, 1930.

Symposium:
An Internist's Approach —

(Continued from page 103)

pancreatitis, should be given very carefully and in small divided doses, for a suddenly produced hypoglycemia is an even more potent stimulus of pancreatic secretion than is hyperglycemia.

Surgical attempts to drain the biliary system are seldom used today and are not recommended. Occasionally, an abscess of the abdominal cavity must be drained. Surgery for known pathological conditions of the gallbladder or biliary tract should be postponed until after complete recovery, but should be performed in order to prevent recurrences.

BOOK REVIEWS

SYNOPSIS OF PATHOLOGY by W. A. D. Anderson, M.A., M.D., F.A.C.P. Paper. Third edition. 783 pages with 331 illustrations and 13 color plates. St. Louis: The C. V. Mosby Company, 1952. $8.00.

The latest revision of this well-known member of The C. V. Mosby Company's synopsis series has incorporated much of the recent advances in the rapidly changing field of pathology. Thus, once again this excellent book has been strengthened and its unique position among texts of pathology reinforced. The book serves exceptionally well as a means of reviewing the field of pathology, providing, clearly and concisely, a complete yet brief approach to this tremendous subject. It does not claim to be a complete text and must never be thought of as such. But, as an outline and quick reference, it seems unsurpassable. The book follows a plan of discussing first the general, then the infectious diseases, the metabolic diseases, and last the pathology of the organ systems. There are many excellent illustrations. In all, this book serves well as a thorough synopsis and is recommended highly for those who seek such a text.

DISEASES OF METABOLISM edited by Garfield G. Duncan, M.D. Cloth. Third Edition. 1179 pages, illustrated. Philadelphia: W. B. Saunders Company, 1952. $15.00.

This text deals exceptionally well with some of the most interesting diseases known to medicine: diseases of porphyrin metabolism, xanthomatoses, diabetes insipidus and mellitus, *etc.* The chapters on carbohydrate, protein, and lipid metabolism are outstanding as are those of "Water balance in health and disease," and "Diseases of the thyroid." In general, the anatomy and physiology of each topic are discussed first, followed by a discussion of the alterations occurring in disease. Diagnosis and therapy are discussed, with emphasis on the recent developments. There are twenty contributors, each of whom has contributed notably well. There are many illustrative cases which give reality to the text, as well as many fine references for those who are inspired to study the subject further. As expected, this revision has served to bring the material up to date and has enabled the editor to shift emphasis to those topics currently of most interest. This book correlates the study of physiology and pathology with clinical medicine in a remarkably interesting and provocative manner, and is highly recommended to all of our readers as an outstanding text.

HUMAN BIOCHEMISTRY by Israel S. Kleiner, Ph.D. Cloth. Third Edition. 695 pages with 83 illustrations and 5 color plates. St. Louis: The C. V. Mosby Company, 1951. $7.00.

This well-known text has been revised in order to keep up with the many recent advances in the field of biochemistry. The discussions on such topics as mineral metabolism and water balance, respiration and acid-base balance, blood coagulation, and vitamin A function have been altered and expanded. In rereading this fine text, once again we have marveled at the clarity of the author and the ability he has to make the material, though often complex, easily understood. The emphasis is on *applied* biochemistry, which gives added life to the book. The text is very well designed for the beginning and clinical student and the general practitioner who wishes an excellent treatise on the biochemical functions of the human being in health and disease. Key references, both general and specific, at the end of each chapter add to the book's value. Once again, this text is highly recommended to all.

PRACTICAL DERMATOLOGY by George M. Lewis, M.D., F.A.C.P. Cloth. First Edition. 328 pages with 405 illustrations on 99 figures. Philadelphia: W. B. Saunders Company, 1952. $7.50.

The phrase below the title of this book is ". . . for medical students and general practitioners" and the book is very neatly designed and written to fulfill that purpose. The author, in recognition of the fact that dermatology is often a maze of perplexities to the general physician and student, tends to keep his text at a very practical and useful level. After an introduction which describes the diagnostic methods and terminology of the subject, the author proceeds systematically to describe the disease entities with which his specialty is concerned. The symptoms, diagnosis, etiology, and treatment are fully described, and are usually reinforced with many illustrations clearly reproduced though, unfortunately, not in color. The book is completed with a fine chapter on dermatologic therapy in general, a formulary, and important references. It is highly recommended to those for whom it was written.

NUTRITION IN HEALTH AND DISEASE by James S. McLester, M.D. and William J. Darby, M.D., Ph.D. Cloth. Sixth Edition. 710 pages with 14 figures and 145 tables. Philadelphia: W. B. Saunders Company, 1952. $10.00.

This well-known text is by no means a mere handbook for physicians in regard to nutritional needs in health and disease, but an excellent, thorough, and quite current volume devoted to the physiology and biochemistry of nutrition and their application to the practice of medicine. The first part of the text provides an outstanding and thorough discussion of normal utilization of food, vitamins, and inorganic nutrients; a discussion of food products; and a study of the diet in health. Each section is smoothly written, highly informative, and quite complete — with many current references at the end of each chapter. The second section covers the nutritional aspects of all fields of medicine. The material is again well written, very complete, and of considerable value. To illustrate the diversity of the material, there can be found chapters on infant feeding, diseases of all organ systems, food poisoning and allergy, nutrition in surgery and industry, *etc.* A large appendix, containing a great deal of information concerning food products and many useful tables rounds out this text to make it one of great value for any student or practitioner.

CARDIOGRAPHY IN GENERAL PRACTICE by Abraham I. Schaffer, M.D. Cloth. First Edition. 135 pages with 55 illustrations. Baltimore: The Williams & Wilkins Co., 1952. $3.00.

This book is specifically designed to provide students and practitioners with a short introduction into the principles and basic features of electro-cardiography. The chapters are brief and concise and include discussions of the fundamental electric activity of the heart and the various clinical entities which are diagnosed by electrocardiography (such as dysrhythmias, infarction, pericarditis, cor pulmonale, *etc.*), vectorcardiography, and ballistocardiography. There is a valuable glossary of terms at the end of the book. The material is simply written with an emphasis on fundamentals. The illustrations are neat and include many electrocardiographic, vectocardiographic, and ballistocardiographic tracings. Of particular value are the two chapters on the electrical aspects of the heart and the electrocardiogram. The text is a very handy work for general practitioners and students, and offers all a summary of the most recent developments in the science of cardiography.

CORRELATIVE CARDIOLOGY by Carl F. Shaffer, M.D., F.A.C.P., and Don W. Chapman, M.D., F.A.C.P. Cloth. First Edition. 525 pages. Philadelphia: W. B. Saunders Co., 1952. $9.50.

The subtitle of this book is "An integration of cardiac function and the management of cardiac disease," but this hardly describes the scope or form of the text adequately. This book is written in outline form with a simplicity of style that enables the 525 pages of the book to contain a great deal of useful information. Its early chapters outline such fundamentals in the study of heart disease as anatomy, embryology, physiology, history taking, physical diagnosis, diagnostic procedures, *etc.* Later chapters discuss specific disease entities using the principles established in earlier chapters. The book makes a unique handbook for anyone interested in cardiology or diseases of the heart in general. It does not attempt to be thorough, but does give the reader a well written outline of almost all the problems in the field. It is thus a handy book to use as a means of approaching cardiology as well as a ready reference or review book for the student or practitioner who does not have the time to refer to more comprehensive volumes.

HANDBOOK OF ORTHOPAEDIC SURGERY by Alfred Rives Shands, Jr., B.A., M.D. Cloth. Fourth Edition. 644 pages with 192 illustrations. St. Louis: The C. V. Mosby Co., 1952. $8.00.

With the collaboration of Dr. Richard Beverly Raney of the University of North Carolina, Dr. Shands has produced a fine revision of this text. This handbook, which is designed specifically for the medical student and general practitioner, has remained a concise book which emphasizes basic principles. It is an ideal compromise between the superficial outline form and the absolutely thorough text. Its bibliography has been revised up to March, 1952, an important feature in these days of constant change. New drawings have been added and, for the first time, these are now supplemented with seventy-nine roentgenograms which have been reproduced rather well. Ten new short sections, on such topics as Brucellosis, Gout, The Foot of the Normal Child, and Morquio's Disease, have contributed to the thoroughness with which all topics in the field are considered. The material itself is clearly written and easy to understand. This revision has served to improve both the written matter and the illustrations, enabling this handbook to remain as one of the outstanding fundamental works in its field.

DISEASES OF THE ENDOCRINE GLANDS by Louis J. Soffer, M.D., F.A.C.P. Cloth. First Edition. 112 pages with 88 illustrations and 3 colored plates. Philadelphia: Lea & Febiger, 1951. $15.00.

This volume is an extremely well-written and thorough work. Dr. Soffer, a well-known endocrinologist from The Mount Sinai Hospital of New York has been aided by three of his colleagues: Dr. J. Lester Gabrilove, Dr. Henry Dolger (who wrote the section on Carbohydrate Metabolism and Diabetes Mellitus), and Dr. Arthur R. Sohval (who wrote the section on the gonads). With remarkable thoroughness, each of the endocrine glands are discussed; first, from the aspect of the basic sciences, and second, from the aspect of the clinical entities which occur in abnormal function (including the symptoms, diagnosis, work-up, therapy, prognosis, and other pertinent aspects). The book has many other assets, such as the appendix of laboratory tests of endocrine function, excellent illustrations, and a vast, selected bibliography at the end of each chapter. It is difficult to imagine a more complete or better written text in this field, and the book is highly recommended to the general physician, advanced student, and internist alike.

BIOCHEMISTRY AND HUMAN METABOLISM by Burnham S. Walker, M.D., Ph.D.; William C. Boyd, Ph.D.; and Isaac Asimov, Ph.D. Cloth. First Edition. 812 pages with 21 figures and 48 tables. Baltimore: The Williams & Wilkins Co., 1952. $9.00.

As the title of this text indicates, this book concerns itself with human biochemistry and it has sacrificed much organic chemistry for the sake of clinical applications. Such an approach is quite welcome and proves very stimulating and successful. All phases of human biochemistry are well covered and the material is very clearly written. Many important references are supplied. The text does not deal with its material as final, but indicates the unsettled nature of many aspects, emphasizing prevailing concepts. Many sections are excellent. The section on growth which includes the study of nucleoproteins and the biochemistry of cancer is perhaps one of the most interesting. Another noteworthy point is that this text may be easily read by a general practitioner or student who wishes to undertake a home-course in biochemistry without the aid of class instruction. The logic and sequence of the book are easily followed. The textbook is an excellent and practical work which is useful for all.

ABSTRACTS SECTION

ATLAS, DONALD H. (*Assoc. Prof. of Med.*) and MARTIN M. KIRSHEN (*Assoc. Prof. of Med.*). Cirrhosis of the Liver—natural history of the disease. Med. Clin. of N. Amer. 37 (1). Jan., 1953.

The natural history of cirrhosis was reviewed from the early theories of Laennec to the current concepts of the disease. The general term "cirrhosis" will be used to signify the fibrosis following acute or chronic injury to the various components of the liver. An etiologic and morphologic classication of cirrhosis is presented. The clinical aspects of the various types of cirrhosis are discussed. The sequence of events beginning with the diverse types of initial hepatic injury was traced to the end stages of the various classes of cirrhosis. The patho-physiology of the progressive phases of cirrhosis was reviewed and the symptomatology, in its dynamic sequence, was considered.

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BRILL, H. M. (*Asst. Prof. of Gyne. and Obs.*) and MAURICE J. GOLDEN (*Asst. in Gyne.*). Vaginal Hysterectomy—The treatment of choice in benign enlargements of the uterus. Amer. Jour. of Ob. and Gyne. *62* (3). 1950.

1121 vaginal hysterectomy case records were reviewed. Single or multiple fibroids up to and including the size of a five month pregnancy were the principle pathologic conditions encountered, whereas menorrhagia, abdominal pain, and prolapse of the uterus were the chief symptoms presented. Inflammatory disease of the pelvic organs and previous pelvic surgery were not considered contraindications to vaginal surgery, while some relaxation of the vaginal supporting structures and uterine mobility were deemed the necessary requirements for surgery through the vagina. Other surgical procedures may be carried out at the time of the vaginal surgery. The authors believe that the old indications and contraindations to vaginal surgery must be revised. All gynecologists should be adept in vaginal surgery.

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ELIAS, HANS (*Asst. Prof. of Anat.*) and D. PETTY. The Hepatic Artery in Man. Anat. Rec., v. 112, no. 2, February. Paper presented at the American Association of Anatomists, Providence, R. I., March 19-21, 1952.

The portal and hepatic veins establish the internal anatomy of the liver. In the region of the main trunks of the portal vein, ducts and arteries show great variations.

Slightly distally from the bases of the rami venae portae ducts and arteries begin to be associated with the portal branches. The artery forms the well known periductal plexuses, two layered around the medium sized and large ducts. Arterial capillaries, incorporated into the periductal plexuses anastomose with distributing portal veins. All arterio-portal anastomoses observed were associated with these plexuses. Some arterial blood can, thus, reach the paraportal sinusoids through inlet venules.

Arterioles and arterial capillaries located in the portal canals give rise to intralobular arterioles and arterial capillaries. Some of these discharge directly into paraportal sinusoids. More of them run within the hepatic lacunae alongside the sinusoids for varying distances. Such intralobular arterioles empty at all levels into the intralobular sinusoids (not only into the centrolobular sinusoids as the senior author assumed originally). Thus, arterial blood is added to the portal blood at all levels.

The problem was re-investigated because the author found a fallacy in one of his previously employed methods. The present study based on India ink and Berlin blue injections, confirmed the former results, showing, however, a richer intralobular, arterial blood supply than he had formerly assumed to exist.

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FOA, PIERO P., L. SANTAMARIA, H. R. WEINSTEIN, S. BERGER, and JAY A. SMITH (*Dept. of Physiol. and Pharm.*) Secretion of the Hyperglemic-Glycogenolytic Factor in Normal Dogs. Amer. Jour. Physiol., *171* (1): 32-36. Oct., 1952.

Experiments in normal and depancreatized dogs strengthened the hypothesis that the Hyperglycemic-Glycogenolytic Factor is a second hormone normally secreted by the pancreas. The hypothesis was further investigated by means of cross-circulation experiments anastomosing the pancreatoduodenal vein of a donor dog, D, with a femoral vein of a recipient dog, R. The results of these experiments showed that the injection of insulin in dog D was followed by hyperglycemia in dog R suggesting that the pancreas of dog D secreted HGF in response to insulin hypoglycemia. The injection of HGF into dog D was followed by hypoglycemia in dog R suggesting that the pancreas of dog D secreted insulin in response to HGF hyperglycemia. It is suggested that HGF is a second pancreatic hormone and that its secretion, like that of insulin, is regulated by the blood sugar concentration.

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KAMIN, HERMAN N. (*Asst. Prof. of Med.*), HERBERT S. LAKIN (*Assoc. in G-U Surg.*), GERSCHEN L. SCHAEFER (*Class of 1950*), and MILTON GOLDIN (*Dept. of Bact.*). Typhus Fever. Ill. Med. Jour. *101* (6). June, 1952.

A case of Brill's disease in Illinois was reported. The authors stressed, as others have in the past, that a negative Weil-Felix reaction should not exclude the diagnosis, although this case had a marked positive reaction. With the influx of many foreign-born displaced persons and with the return of members of the Armed forces from areas where typhus is epidemic, it behooves us to be on the alert for Brill's disease. Not only active cases, but those harboring a latent disease may act as human reservoirs for further spread of typhus.

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KOENIG, H. (Asst. Prof. Anat.), STAHLECKER, H., and KOENIG, R. S. The Neuromuscular Mechanism of Alkalotic and Hypocalcemic Tetany. Proc. Soc. Exper. Biol. and Med., Vol. 79, 1952.

Tetany was produced acutely in eleven cats and one dog with solutions of sodium bicarbonate, sodium carbonate and a phosphate buffer administered by venoclysis. Fasciculations and tonic spasms involving especially the head and forelimb musculature characterized this form of tetany. Clonic spasms were not seen in anesthetized animals. Section of the hypoglossal nerve did not significantly influence the fasciculations of the tongue musculature. Section of the brachial plexus did not significantly influence the fasciculations or tonic spasms of the forelimb musculature. Paralytic doses of d-tubocurarine blocked all motor manifestations of tetany. It was concluded that all of the motor phenomena characterizing alkalotic and hypocalcemic tetany could result from the spontaneous iterative discharges occurring in peripheral motor nerve fibers or their terminations.

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LEVINE, SEYMOUR *(Alumnus 1947), et al.* Infrared Spectrophotometry of Enteric Bacteria. J. Bacteriology, *65*:10. Jan., 1953.

Infrared spectra of bacteria were obtained using dried films on silver chloride disks. Strong absorption bands at 6.05 and 6.45 μ are ascribed to the peptide linkage of proteins. A band at 8.0-8.1 μ decreases in intensity as a culture ages and can be completely removed by extraction of the cells with dilute alkali or hot trichloracetic acid. These observations suggest that nucleic acid may cause this absorption. Polysaccharides are partly responsible for a broad, deep absorption band at 8.6-10 μ as indicated by the fact that polysaccharides isolated from bacteria by a variety of methods absorb strongly in this range.

Small differences are observed in the spectra of *Aerobacter, Escherichia, Salmonella,* and *Shigella* species. The composition of the growth medium influences the bacterial spectrum. The addition of fermentable carbohydrate to a basic nutrient agar results in characteristic alterations of the bacterial spectra which appear at varying rates in different species. Since these spectral changes do not appear after the addition of a non-utilizable sugar (e.g., lactose in the case of *Salmonella),* they must be related to the metabolism of the carbohydrate.

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LUISADA, ALDO A. *(Assoc. Prof. of Med., Director of Cardiology).* Electrokymography. Proceedings of the First Conference, 1950, U.S.P.H.S. Public. 59. Atrial Phenomena.

Electrokymography lends itself to the study of border pulsation of either the right or the left atrium. Technical details are given. Densograms can be recorded also in the case of the left atrium.

Normal tracings present a negative wave in presystole and a second negative wave in systole. The causes of these waves are discussed.

Auricular fibrillation, auricular flutter, bradycardia with A-V block, and premature contractions are revealed by typical changes of the atrial tracings.

Hypertrophy of one of the atria is revealed by increased depth of the presystolic wave. This wave, on the contrary, becomes small in cases with stenosis of the mitral valve.

A typical pattern revealing "systolic swelling" of the atrium is present in cases with regurgitation from one of the ventricles into the respective atrium.

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MADONICK, M. J., and J. MARGOLIS. *(Alumnus 1949)* Protein Content in Spinal Fluid in Diabetes Mellitus. A.M.A. Archives of Neurology and Psychiatry. *68*. pp. 641-644. November, 1952.

Since the spinal-fluid protein content is at times increased in various diseases of metabolism or disorders in which the metabolism is disturbed, and since diabetes mellitus is an excellent example of a metabolic disorder, cell counts and total protein determinations were made on the spinal fluids of one hundred diabetic patients. Of the one hundred patients, eighty had no objective signs of neurologic involvement. Of twenty with neurologic signs, fifteen had hemiplegia, four, peripherial neuropathy, and one, myelopathy on a diabetic basis.

The spinal fluid of only one patient had a white cell count of five polymorphonuclear leukocytes per cubic millimeter; this patient had no neurologic signs.

The spinal fluid protein value was 50 mg. or more per 100 cc. in only five of the one hundred patients. Four of the five had peripheral neuropathy and the fifth had a recent hemiplegia.

Although there was no relation between the increase in the spinal fluid protein and the duration of the diabetes, there was a suggestive relation between increase in the spinal fluid protein and the severity or the difficulty in control of the diabetes.

No relation between the spinal fluid protein and the blood sugar level was demonstrated. No relation of the spinal fluid to the serum protein concentrations was observed in the forty-five patients so tested.

Our results showed that an increase in the cerebrospinal fluid protein is found in diabetes mellitus only when neurologic complications are present. The commonest complication with an increase in the spinal fluid protein concentration was peripheral neuropathy.

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STEVENSON, HEBER J. R. and SEYMOUR LEVINE. *(Alumnus 1947)* Infrared Spectra of Pneumococcal Polysaccharides. Science, *116*: 705. Dec. 26, 1952.

Infrared spectra of type-specific immunopolysaccharides derived from the capsules of pneumococci were obtained using a Perkin-Elmer double-beam recording spectrophotometer. The spectra of all types examined were clearly distinguishable. Certain spectral relationships paralleled known chemical similarities. Polysaccharides of the same type prepared by different methods were found to have very similar spectra. These results indicate that infrared spectrophotometry may be useful in the identification of type-specific immunopolysaccharides, as a criterion of purity, and in elucidating chemical structure.

SCHOOL NOTES AND NEWS

NEW APPOINTMENTS TO EDITORIAL BOARD

The *QUARTERLY* takes great pride in announcing the appointments by President John J. Sheinin of Doctors Emanuel Marcus and Harold Koenig to the Editorial Board of the *QUARTERLY*. Both Dr. Marcus and Dr. Koenig have been extremely prolific as authors of scientific articles, and have written for the *QUARTERLY* as well as many other publications.

Dr. Koenig was graduated from The Chicago Medical School, Class of 1946, after having received the degree of Bachelor of Science in Biological Science from Rutgers University in 1942. While attending medical school, Dr. Koenig found time to study at the Northwestern University Neurologic Institute, where he received a Master of Science degree in Neurology in 1945. After an internship at the Walther Memorial Hospital of Chicago(1946 to 1947) Dr. Koenig went to Philadelphia where he continued his postgraduate studies in Anatomy and Neurology at the University of Pennsylvania School of Medicine. Concurrently he held the position of Instructor in Microscopic and Neurologic Anatomy at the same school. Following the receipt of the degree of Doctor of Philosophy in Anatomy in 1948, Dr. Koenig was appointed Assistant Professor of Gross Anatomy at the University of Pennsylvania Medical School. He held this position until his return to the The Chicago Medical School in September, 1949, where he was appointed Assistant Professor of Gross Anatomy.

While a student in medical school, Dr. Koenig's ability in Anatomy was recognized and he served as Prosector from 1944 to 1947. Extracurricular activities while in school included membership in Phi Lambda Kappa Fraternity. Other societies in which he holds membership are Phi Beta Kappa, the American Association for the Advancement of Science, the American Association of Anatomists, the Philadelphia Physiological Society, the Histochemical Society, and Sigma Xi.

Dr. Koenig has written several articles in conjunction with his wife, who, incidentally, is also a graduate of The Chicago Medical School. Dr. Ruth Koenig is at present a Resident in Neuropsychiatry at the Illinois Neuropsychiatric Institute, having recently completed a residency at the Chicago State Hospital.

Dr. Emanuel Marcus is the new representative of the Clinical Staff on the Editorial Board. Dr. Marcus is a resident of Hammond, Indiana, where he is on the staff of Saint Margaret's Hospital. His education was received at the University of Chicago, where he obtained a Bachelor of Science degree in 1934 and a Doctorate of Philosophy in Physiology in 1937. He carried on his studies at the Rush Medical College of the University of Chicago and was granted the degree of Doctor of Medicine in 1942. He took his internship at the Michael Reese Hospital of Chicago from 1942-1943.

While working for his advanced degrees, Dr. Marcus was a Research Associate in the Department of Physiology of the University of Chicago from 1934 to 1937. Other teaching experiences before his appointment, in 1949, to The Chicago Medical School as Instructor in Surgery include a course which he gave at the Maxwell Field Air Force Base, Alabama, on "Recent Advances in Surgery."

Dr. Marcus is affiliated with the staff of Michael Reese Hospital of Chicago. Among his most interesting experiences are those which occurred during his visit to Israel. Many impressions of his stay and service there are related in the October, 1949 issue of the *QUARTERLY*. While there, Dr. Marcus was Chief of Surgery in the Negev and of the Italian Hospital in Haifa and Co-Chairman of the department of Surgery at Tel Litwinsky Hospital.

At present, Dr. Marcus is an Assistant Professor of Clinical Surgery at The Chicago Medical School and is a member of Phi Beta Kappa, Alpha Omega Alpha, the American Medical Association, the Chicago Medical Society, and the Lake County (Indiana) Medical Society. He is a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons and the International College of Surgeons.

In addition to teaching and an active practice, Dr. Marcus has made time to carry out numerous research projects. Among his present studies are several in Cardiovascular Surgery, including surgery of mitral stenosis and heart transplantation. Recent publications by Dr. Marcus include "Parenteral Administration in Surgical Patients," published in the Indiana State Medical Journal, and "Mammalian Heart Transplants," published in the A.M.A. Archives of Surgery.

Again the *QUARTERLY* welcomes to its Editorial Board two gentlemen whose experience and ability will add much to the growth of this publication.

Appointments as Attending Men to the staff of the Cook County Hospital were received by twenty-five members of the faculty of The Chicago Medical School. These positions were awarded on the basis of competitive examinations and service to the hospital. The appointments were effective as of January 1, 1953 and have a tenure of six years. The *QUARTERLY* joins the Administration, Faculty, and student body of The Chicago Medical School in congratulating the following staff members on the receipt of this signal honor:

(New appointments are indicated by an asterisk.)

Contagious Diseases—Dr. Archibald L. Hoyne.

Gynecology—Dr. Egon W. Fischmann

Medicine—Dr. Donald H. Atlas*, Dr. Petter Gaberman, Dr. Harry J. Isaacs, Dr. Abraham Rimmerman, Dr. Meyer J. Steinberg*

Neurology—Dr. Harry H. Garner

Neuro-Surgery—Dr. Milton Tinsley

Obstetrics—Dr. Charles Fields, Dr. Philip J. Stein*

Ophthalmology—Dr. Samuel J. Meyer*

Orthopedic Surgery—Dr. Donald S. Miller

Otolaryngology—Dr. Maurice H. Cottle, Dr. Jack A. Weiss

Pathology—Dr. Benjamin H. Nieman
Pediatrics—Dr. Julius Aronow*, Dr. Maxwell P. Borovsky, Dr. Hyman S. Gordon*, Dr. Benjamin M. Levin.
Surgery—Dr. Earle I. Greene, Dr. Herman A. Jacobson, Dr. Leo N. Zimmerman
Thoracic Surgery—Dr. Saul A. Mackler
Tuberculosis—Dr. David G. Lerner

By virtue of these appointments, curriculum alterations have been made enabling students in the Junior and Senior years to have increasing amounts of clinical material for their studies. These improvements, as well as those gained through the added facilities of the expanding Mount Sinai Hospital Research Foundation mark two great steps forward in the progress of The Chicago Medical School.

FACULTY NEWS

Congratulations to Mr. and Mrs. Milton Geerdes on the birth of their first child, David Kurt, on March 9, 1953. Mr. Geerdes is the School's Registrar.

Department of Medicine

President John J. Sheinin has announced the appointment of Dr. Giorgio Gamna as Research Assistant to Dr. Aldo Luisada, Program Director of Cardiology and Associate Professor of Medicine.

Dr. Gamna was formerly Resident in Medicine at the Torino Medical School Hospital. During his stay here, he will assist Dr. Luisada in his work on phonocardiography.

Dr. Aldo Luisada has been elected Secretary of the Scientific Section of the Chicago Heart Association.

Department of Microbiology and Public Health

Dr. James G. Shaffer, Chairman of the Department of Microbiology and Public Health, has received $10,098 from the United States Public Health Service for "Studies on the Growth Requirements of Endamoeba Histolytica."

Dr. Harold Elishewitz, Assistant Professor of Parasitology, has recently been elected First Vice President of the State Microscopical Society of Illinois.

Department of Neurology and Psychiatry

Dr. Leroy P. Levitt (Class of 1943), Instructor in Psychiatry, has been appointed Consultant in Psychiatry to the Rest Haven Convalescent Home, Chicago, Illinois.

Department of Physiology and Pharmacology

President John J. Sheinin has announced the appointment of Dr. Angelo Fasoli as Research Assistant to Dr. Piero P. Foa, Professor of Physiology and Pharmacology and Associate in Clinical Medicine. Dr. Fasoli is on leave of absence from the University of Milan, where he is Assistant Professor of Medicine.

Department of Obstetrics and Gynecology

The office of President John J. Sheinin has announced the appointments of Doctors Bernard M. Greenwald and Donald J. Sabath as Associates in Obstetrics and Gynecology.

Department of Pathology

Dr. Israel Davidsohn, Professor and Chairman of the Department of Pathology, has received a renewal of a grant of $6,372 from the United States Public Health Service for continuation of his studies in the "Immuno-Hematologic Aspects of Hemolytic Anemia."

Dr. Kurt Stern, Assistant Professor of Pathology, has been appointed Chairman of Scientific Exhibits for the 1953 Convention of the American Association of Blood Banks.

Department of Pediatrics

The office of President John J. Sheinin has announced the appointment of Dr. Marvin P. Padorr as Assistant in Pediatrics.

Congratulations to Dr. Jerome H. Diamond, Assistant in Pediatrics, and the former Miss Sally Moss on their marriage on March 15, 1953.

Department of Surgery

President John J. Sheinin has recently announced the appointment of Dr. Renato Baserga as Research Assistant to Dr. Philippe Shubik, Assistant Professor and Coordinator of Oncology.

The office of President John J. Sheinin has also announced the appointments of Dr. Samuel Nien-Tsu Wong as Instructor in Surgery and Dr. William F. Lichtman as Assistant in Orthopedic Surgery.

EDITOR'S NOTE:

Through recent correspondence, many alumni have expressed their interest in the activities of their former classmates and friends. The QUARTERLY shares this sincere interest but depends mainly upon its readers as a source of information. Alumni are urged to submit data regarding themselves--change of address, hospital staff appointments, personal items, and any other items of interest—to the Editor.

Class of 1932

Dr. Francis X. Graff of Freeport, Illinois, has recently been elected President of the Staff of the Saint Francis Hospital for the current year.

Class of 1935

Dr. Jacob Chalfin recently introduced a resolution before the Industrial Medical Association (formerly the American Association of Industrial Physicians and Surgeons) to the effect that membership certificates suitable for framing be issued to its active members in the United States, Hawaii, and Canada. This was voted on and approved by the Board of Directors of this organization and, for the first time since its founding in 1916, the active members of the Industrial Medical Association will receive such certificates. Many graduates of The Chicago Medical School who are engaged in industrial and railroad medical and surgical work are members of this national organization.

Class of 1943

Congratulations to Dr. and Mrs. Sydney S. Lazarus on the birth of a daughter, Anne Elinor, on November 12, 1952.

Class of 1944

Dr. Herbert A. Berger, after twelve months of active duty in Korea, has recently resumed his practice at 740 East 6th Street, New York City. While serving with the 185th Combat Battalion, Dr. Berger received the Bronze Star Medal as well as several letters of commendation from the Far Eastern Command Surgeon. Dr. Berger is currently affiliated as Assistant in Medicine, Beth Israel Hospital, New York City, and as Assistant in Pediatrics, Greenpoint Hospital, Brooklyn, New York.

Class of 1946

Captain Bernard H. Shulman, a former Editor of the *QUARTERLY,* is now stationed at the United States Army Hospital, Fort Belvoir, Virginia. Before his entrance into the service Dr. Shulman was co-author of an article entitled "Time of Healing of Gastric Ulcers," which appeared in *Gastroenterology,* Vol. 20, No. 1.

Dr. Arnold L. Statsinger, also a former Editor of the *QUARTERLY,* has recently returned from two years of active duty with the United States Army Medical Corps. During that time he was Chief of the Laboratory Service at the United States Army Hospital at Fort Jackson, South Carolina. Dr. Statsinger is now in training at the National Cancer Institute in the Delafield Hospital, New York City, where he is under the direction of Dr. Arthur Purdy Stout. Congratulations are in order to Dr. and Mrs. Statsinger (nee Miriam Joan Walder) on the occasion of their marriage in June, 1952.

Class of 1947

The *QUARTERLY* would like to take this opportunity to thank Dr. Seymour Levine for his recent communication. Dr. Levine, presently serving as Senior Assistant Surgeon, United States Public Health Service, has recently published several articles as a result of his research on infra-red spectrophotometry. This work was completed at the laboratories of the Environmental Health Center of the United States Public Health Service, Cincinnati, Ohio, where Dr. Levine is Associate Chief of the Spectrophotometry Unit of the Bacteriology Department. Abstracts of the aforementioned publications are published elsewhere in this issue.

Dr. Robert R. Simner has announced the opening of an office for the practice of Anesthesiology at 4059B Central Expressway, Dallas, Texas.

Class of 1948

Dr. Harold N. Brown is at present stationed at the Municipal Airport, Wichita, Kansas, after having completed studies at the School of Aviation Medicine at Randolph Field, San Antonio, Texas.

Captain William C. Feldman of Kingston, New York, has been awarded a Meritorious Commendation in behalf of his unit, the 125th Medical Detachment. Dr.

Feldman played a large role in abating a potential smallpox epidemic in Korea. After more than one year of service in Korea, Dr. Feldman has returned to the United States on a rotation of duty.

Class of 1949

The *QUARTERLY* wishes to acknowledge receipt of a recent letter from Lieutenant Jack Margolis, presently stationed at Turner Air Force Base, Albany, Georgia. Congratulations are in order to Dr. and Mrs. Margolis, although somewhat belated, on the occasion of the birth of their son, Robert, on May 2, 1952. Before entering the service, Dr. Margolis was co-author of an article, "Protein Content of the Spinal Fluid in Diabetes Mellitus." This article appeared in the *American Medical Association Archives of Neurology and Psychiatry,* November, 1952. An abstract of this publication is published elsewhere in this issue.

Class of 1950

Congratulations to Dr. Harry L. Rosenthal on the occasion of his marriage to the former Miss Ann Katz on November 3, 1952.

Class of 1951

Dr. Samuel A. Farber takes pleasure in announcing the opening of his office, The Harbor Medical Center, for the general practice of Medicine and Surgery at 525 South Pacific Coast Highway, Redondo Beach, California.

Upon completion of a year's Residency in Pediatrics at the Metropolitan Hospital, New York City, Dr. Arthur Lisbin will be associated with the New York Hospital (Cornell University) as an assistant Pediatrician to the Outpatient Department and as Clinical Fellow in Pediatrics.

Congratulations to Dr. and Mrs. Louis Kolokoff on the birth of a daughter, Valerie, on February 14, Valentine's Day, 1953.

Class of 1952

Dr. and Mrs. Marvin Markowitz proudly announce the birth of a son, Nathan Alan, on January 12, 1953.

The *QUARTERLY* would like to take this opportunity to congratulate the following members of the Class of 1952 on their appointments to the following Residencies:

Dr. Norman H. Blass—Chicago Lying-In Hospital, Chicago, Illinois—Obstetrics and Gynecology.

Dr. Sanford I. Cohen—Colorado General Hospital, Denver, Colorado—Psychiatry.

Dr. Eugene H. Fierer—Brooklyn Jewish Hospital, Brooklyn, New York.—Medicine.

Dr. Abraham E. Goldminz—Kingsbridge Veterans Administration Hospital, Bronx, New York—Medicine.

Dr. Peter H. Griesbach—Queens General Hospital, Jamaica, New York—Pediatrics.

Dr. Mark Josel—Queens General Hospital, Jamaica, New York—Medicine.

Dr. Herbert S. Kaiser—Coral Gables Hospital, Coral Gables, Florida—Surgery.

Dr. Walter Kitt—Illinois Neuropsychiatric Institute, Chicago, Illinois—Psychiatry.

Dr. Burton M. Krimmer—Michael Reese Hospital, Chicago, Illinois—Medicine.

Dr. Montague Lane—Brooklyn Jewish Hospital, Brooklyn, New York—Medicine.

Dr. Maurice H. Laszlo—Brooklyn Jewish Hospital, Brooklyn, New York—Medicine.

Dr. Edmon B. Lee—Queens General Hospital, Jamaica, New York—Medicine.

Dr. Marvin Markowitz—Beth David Hospital, New York, New York—Medicine.

Dr. Murray Neustadter—Bellevue Hospital, New York, New York—Obstetrics and Gynecology.

Dr. Lloyd J. Paul—Harper Hospital, Detroit, Michigan—Medicine.

Dr. Harold Ratner—Brooklyn Jewish Hospital, Brooklyn, New York—Pediatrics.

Dr. Murray K. Rosenberg—Brooklyn Jewish Hospital, Brooklyn, New York —Anesthesiology.

Dr. Richard Sabransky—Mount Sinai Hospital, Chicago, Illinois—Surgery.

Dr. Robert L. Schrier—New York Hospital, New York, New York—Anesthesiology.

Dr. Lester Schwartz, Hines Veterans Administration Hospital, Hines, Illinois—Anesthesiology.

STUDENT NEWS

Senior Class

We wish to congratulate Marvin Kranis of New York City and the former Miss Elaine Frank of Brooklyn, New York, on their marriage on March 22, 1953.

Congratulations to Martin J. Rubinstein of Pittsburgh, Pennsylvania, on his marriage to the former Miss Bonnie Paull of Minneapolis, Minnesota, on March 22, 1953.

Congratulations to Lesther Winkler of Pittsburgh, Pennsylvania, and the former Miss Charlotte Siedband of Chicago, Illinois, on the occasion of their marriage on March 29, 1953.

Best wishes to Walter Griesbach of Flushing, New York, on his recent engagement to Miss Janet Simon of the Bronx, New York.

Junior Class

Congratulations to Gene and Dottie Trachtenberg on the birth of their first child, Marcia Lynn, on December 15, 1952.

Best wishes to Melvin Post of Brooklyn, New York, and the former Miss Pearl Rae Godow of Chicago, Illinois, on their marriage on March 22, 1953.

Congratulations to Arnold Adicoff of the Bronx, New York, on his recent engagement to Miss Ruth Kahn of Milwaukee, Wisconsin.

Sophomore Class

Congratulations to Seymour and Leila Stricker on the birth of their first child, Jeffry Bart, on December 14, 1952.

Congratulations to Chuck and Suki Weisenthal on the birth of their first child, Harriet Sue, on February 22, 1953.

Congratulations to Stuart Eichenfield of Brooklyn, New York, and the former Miss Frances Fassler of Brooklyn, New York, on their marriage on December 30, 1952.

Best wishes to Ernest Weitz of New York City, and the former Miss Renee Login of Chicago, Illinois, on the occasion of their marriage on February 7, 1953.

Best wishes to J. Arthur Brandt of Brooklyn, New York, on his engagement to Miss Anne Holmelund of Geneva, New York.

Freshman Class

Congratulations to Howard Rose of Miami Beach, Florida, on his marriage to the former Miss Muriel Joan Meyer of Chicago, Illinois, on December 27, 1952.

Best wishes to Myles Marks of Hicksville, Long Island, on his engagement to Miss Sandra Lomench of North Bellmore, Long Island, on December 29, 1952.

ORGANIZATION NEWS

Student Council

The Student Council has continued to pursue its active program to satisfy the social and educational needs of the student body. New plans are constantly under discussion and new programs are constantly being formulated so that the Student Council can steadily increase its service to the students.

The "Second Annual Spring Dance" sponsored by the Student Council is scheduled to be held April 25, 1953 at the Hamilton Hotel. This dance, which was a tremendous success last year, promises to surpass all previous school-wide dances. The "Extra-Added Attraction" will be an entertainment program prepared by the Sophomore Entertainment Committee under the direction of Mel Greenblatt.

An experiment by the council to provide non-medical educational films has met with an excellent reception by the student body. These films, presented at noon on Mondays in the "Amph. A Playhouse," have dealt with many diverse subjects ranging from sculpture to conservation programs. The council plans to continue this film series and will attempt to maintain and improve the caliber of the films shown.

A permanent committee has been established to publish the *Foramen*. This committee, headed by Mort Lacher, hopes to publish the *Foramen* on a regular schedule, enlarge its size, improve its contents, and create regular features. It is hoped that, in this way, the *Foramen* will develop into the voice of the student body.

Phi Lambda Kappa

On Sunday afternoon, January 18, 1953, the Alpha Rho chapter of Phi Lambda Kappa held its Annual Induction Dinner at the Sheridan Plaza Hotel. The new members were inducted in an impressive ceremony which was ably conducted by President Kenneth Cohen.

At the first meeting of last quarter, our delegates to the National Convention which was held December 29-31 at Detroit, Michigan, reported to the chapter. The main result of the convention was a change in the constitution making any medical student, regardless of his race or religion, eligible for membership in Phi Lambda Kappa.

Alpho Rho Chapter in conjunction with the Phi Lambda Kappa National Organization has begun a "Books for Israel" drive. Any medical books which you can donate will be accepted and sent to the Hebrew University Medical School in Israel. We appeal to everyone to cooperate with us in this worthy endeavor.

Playing its usual leading role in the Phi Lambda Kappa Educational Program is the forthcoming Fourth Annual Maurice Oppenheim Memorial Lectureship. The chapter was fortunate to obtain this year Dr. Sidney Farber, Director of Research of the Children's Cancer Research Foundation of Boston, who will speak April 10, 1953 at Kling Auditorium of the Mount Sinai Hospital of Chicago.

Phi Delta Epsilon

Frater Sandy Kaplan is to be commended for bringing to the entire student body of The Chicago Medical School, on behalf of Phi Delta Epsilon, a series of lectures on non-medical topics. The series commenced on January 23rd, with a talk by Dr. Charles Kaplan of the Department of English at Roosevelt College.

The bi-weekly series of films on medical topics has met with an unqualified success, which has gratified the Beta Tau chapter. This film series will be continued until the end of the spring quarter.

The lighter side of fraternity life has not been neglected either. The annual all-city dinner-dance was held on February 21st at the Furniture Club of Chicago. At that time, the men making up the record-breaking pledge class of the chapter were officially inducted into the fraternity.

Association of Internes and Medical Students

The tempo for the coming year was set at the Christmas Convention of the Association of Interns and Medical Students held at the University of Chicago. The educational highlights of the convention were a seminar on cardiology by Drs. Aldo A. Luisada and Louis Katz and a dissertation on the structure and function of the cerebral cortex by Dr. Percival Bailey. Panel discussions were held on the subjects of Academic Freedom, Discrimination in Medicine, The Doctor Draft, and Medical Economics. It was resolved that A.I.M.S. would continue in its endeavors to help solve the social and socio-economic problems facing the medical profession.

The local chapter, with the aid of its faculty advisor, Dr. Piero P. Foa, has planned a lecture series entitled, "The Internship, Then What?" The lectures will attempt to acquaint the student and intern with the advantages and disadvantages of specialty practice as compared with general practice.

Student American Medical Association

The Student American Medical Association of The Chicago Medical School has continued its expanding program of providing educational lectures for the students. Early in January, Dr. Warren Cole gave a very interesting talk on "Diseases of the Gall Bladder." The remainder of the schedule was as follows:

January 30—Dr. Mathew Taubenhaus, "The Modern Concept of Collagen Diseases."

February 3—Dr. I. Snapper, "The Pathogenesis and Treatment of Acute Anuria."

February 27—Dr. Hans Popper, "The Differential Diagnosis of Jaundice by Laboratory Tests."

March 13—Dr. Geza de Takats, "Problems in Hypertension."

Elections were held early in January and the new officers took their posts in March. The newly elected officers are: President, Herbert Sohn; Vice-President, Helmuth Stahlecker; Secretary, Lawrence Strenger; and Treasurer, Bernard Deitch.

THE CHICAGO MEDICAL SCHOOL

QUARTERLY

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Chicago 12, Illinois

The Chicago Medical School *QUARTERLY* is published four times yearly by The Chicago Medical School for the dissemination of current medical news and for the advancement of medical science with a student staff under the supervision of a faculty editorial board.

Instructions to Contributors

Articles must be typewritten, double spaced, and the original copy submitted.

All articles are accepted on the condition that they are contributed solely to this publication.

A minimum number of illustrations will be furnished by the *QUARTERLY* provided the photographs or drawings are of suitable quality.

Reprints will be furnished by the *QUARTERLY* without charge and must be requested when the manuscript is submitted.

Manuscripts for publication should be addressed to The Editor, The Chicago Medical School *QUARTERLY*, 710 S. Wolcott Avenue, Chicago 12, Illinois.

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Bibliographies must conform in style to that used in *Quarterly Cumulative Index Medicus.*

Permission must be obtained from the *QUARTERLY* for use of all or part of any articles in this publication. Permission will usually be granted provided proper credit is given.

LECTURE SERIES

The Chicago Medical School — Tuesdays, 12:30 P. M.

710 South Wolcott Avenue — Amphitheater A

PROGRESS IN ENDOCRINOLOGY

1953

APRIL 7 — The Mechanism of Insulin Action, a Round Table Discussion[1].

Dr. Sidney P. Colowick, Associate Professor of Biology, McCollum Pratt Institute, Johns Hopkins University, Baltimore.

Dr. Rachmiel Levine, Chairman, Department of Medicine, Michael Reese Hospital.

Dr. DeWitt Stetten, Jr., Chief, Division of Nutrition and Physiology, Public Health Research Institute of the City of New York, Inc.

APRIL 14 — Parathyroid Hormone and Calcium Metabolism.

Dr. I. Snapper, Director of Medical Education, Cook County Hospital, and Professor of Medicine, The Chicago Medical School.

APRIL 21 — Glucagon: the Hyperglycemic Hormone of the Pancreas.

Dr. Piero P. Foa, Professor of Physiology and Pharmacology, The Chicago Medical School.

MAY 5 — The Liver in Endocrine Disturbances.

Dr. I. J. Pincus, Associate Professor of Physiology, Jefferson Medical College, Philadelphia.

MAY 12 — The Use of Radioiodine in Thyroid Disease.

Dr. Dwight E. Clark, Professor of Surgery, University of Chicago.

MAY 19 — Adrenal Glands and Cancer of the Breast.

Dr. Thomas L. Dao, Instructor, Ben May Cancer Research Laboratory, and Department of Surgery, University of Chicago.

MAY 26 — Adrenal Cortical Steroids in the Treatment of Rheumatic Diseases.

Dr. Edward F. Rosenberg, Assistant Professor of Medicine, The Chicago Medical School, and Attending Physician, Michael Reese Hospital.

JUNE 2 — Hormones of the Gastro-intestinal Tract[2].

Dr. Morton J. Grossman, Professor of Clinical Science, University of Illinois, and Chief, Clinical Investigation Division, Medical Nutrition Laboratory, United States Army.

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1. Sponsored by the Student American Medical Association.
2. Sponsored by the Association of Internes and Medical Students.